The Journal of Thoracic Surgery

VOL. 7 JUNE, 1938 NO. 5

Original Communications

TREATMENT OF ACUTE SUPPURATIVE PLEURITIS FOLLOWING RUPTURE OF LUNG ABSCESS

FRANK S. DOLLEY, M.D., AND JOHN C. JONES, M.D.
LOS ANGELES, CALIF.

ENCAPSULATED empyema that develops by pleural extension from a contiguous lung abscess, or generalized empyema, due to secondary infection of a pleural effusion, the result of necrosis in the lung beneath, usually responds to the recognized methods of handling empyemas in general and runs a comparatively benign course. Acute suppurative pleuritis, however, that occurs with almost overwhelming suddenness by direct rupture of a lung abscess through the visceral pleura is a vastly different affection. A discussion of a treatment for this last named condition is the purpose of this paper.

The pleural shock from sudden rupture of the lung abscess in conjunction with the exquisite intercostal pain that occurs from parietal pleural involvement is often extreme. Not rarely the illness has been interpreted as due to coronary occlusion and for many precious hours has been treated as such. The pleuritic pain greatly limits the depth of respiration. Marked dyspnea is immediately a prominent symptom. The patient almost at once seems overwhelmed. The lung is more and more compressed both by air trapped out in the pleural space through the patent bronchi in the abscess walls and by the copious serous effusion issuing from the pleural surfaces. At the same time the mediastinum steadily shifts to the opposite side at the expense of the contralateral lung which, with all the aid the heart can furnish it, is already trying desperately to supply the rapidly increasing cerebral and circulatory appeal for more oxygen. The culminating burden that cannot long

Read before the Twentieth Annual Meeting of the American Association for Thoracic Surgery at Saranac Lake, N. Y., May 31, June 1 and 2, 1937.

be supported is the development of toxemia secondary to the pleuritis that insidiously makes more and more insistent demands for the little oxygen that remains available.

Sergent and Iselin[1] in their paper on pleural effusions with lung abscess, delivered before this Society in 1933, limit their consideration of acute suppurative pleuritis or "pleural phlegmon" as they term it, to these words: pleural phlegmon "we shall not discuss; the classical variety represents an almost invariably fatal complication." The experience of American surgeons in dealing with this condition has been more fortunate. Neuhof and Hirshfeld[2] in their excellent publication on the diagnosis and treatment of so-called putrid empyema made very emphatic the lifesaving importance of immediate and wide open drain-

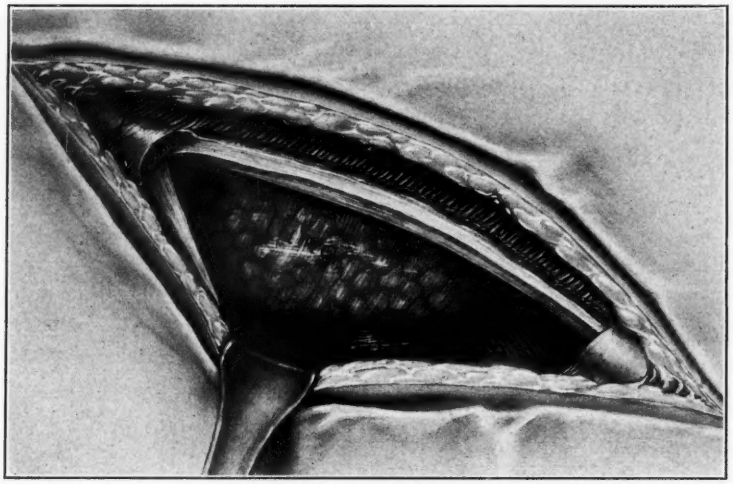


Fig. 1.—Wide exposure insuring complete dependence even following rise of diaphragm.

age of the pleural fluid secondary to ruptured lung abscess. The most successful of us, however, still finds high mortality.

The situation even before the advent of toxemia is one of extreme emergency. But it is inevitable that the serious mechanical distress of the first few hours will soon be fearfully enhanced by the suppurative pleuritis that is, almost from the time of rupture, steadily but inexorably advancing.

The earlier that immediate and complete evacuation of this highly infectious necrosis-producing fluid is accomplished and some means is taken to prevent its reaccumulation, the greater will be the percentage of recoveries. The time element is even more important than with the notoriously serious ruptured gastric ulcer. Rib resection and large tube drainage meet these requirements, and yet uniformly result in death

unless the thoracotomy opening is maintained airtight or nearly so. The mediastinum has had no time to stiffen, and, as Graham[3] proved years ago, not only has the homolateral lung been still further collapsed but so has the contralateral one, as the mediastinum in inspiration shifts to the opposite side. This increased reduction in vital capacity through the sucking thoracotomy wound is not borne.

Aspiration by needle is but a makeshift in these cases. Immediate, complete, and permanent pleural emptying is essential. Moreover, cellulitis of the chest wall following needle aspiration, despite all precautions to prevent its occurrence, is very frequent with this foul, often anaerobic, infection. Airtight intercostal tube drainage does not afford an opening sufficient to drain such material adequately and with com-

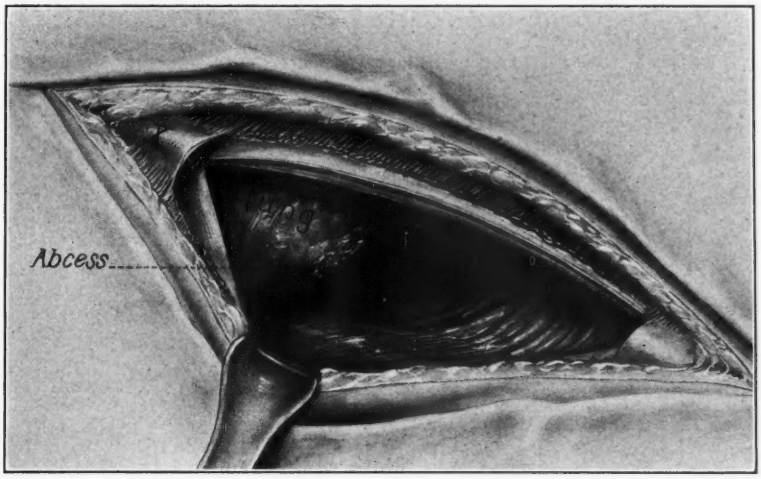


Fig. 2.—Adequate exposure of site of abscess rupture permitted by this incision.

plete dependence. In addition, if inadvertently the tube becomes kinked or occluded, the pus, even the air from the pleural cavity as the patient coughs or strains, may enter the soft tissues deep to the fascia. The infection under the deep fascia spreads with appalling swiftness and is almost invariably fatal.

In our attempt to unite these two prime requirements, namely, (1) immediate wide open and dependent drainage and (2) maximal elimination of inspiratory suction at the drainage opening, we adopted the following simple procedure: through a long curvilinear incision four or five inches of the rib which is found to be at the very bottom of the pleural mixture of abscess contents, pleural effusion, and air, is resected at the lateral border of the erector spinae muscles. The rib resection thus is from a point close to the vertebral transverse process laterally nearly to the posterior axillary line. The parietal pleura is slit wide

open throughout the extent of the rib resection. The depressed diaphragm is on a level with the distal end of the parietal pleural opening. As the diaphragm rises following the removal of the fluid, the thoracotomy opening, which follows the diagonally upward course of the rib removed, still maintains the level of complete dependence even to the top of the diaphragmatic dome.

We feel that it is of vital importance not to attempt to enlarge the opening in the peripheral wall of the lung abscess itself at the time of the pleural drainage. The patient can withstand but a minimal amount of violence at this period. It is absolutely essential that at the earliest possible moment the sucking wound of the chest wall be closed. As Eggers[4] has emphasized, moreover, the majority of lung abscesses that rupture through the visceral pleura recover if the pleuritis itself is sustained. In one of our cases, three days following the above mentioned

Fig. 3.—Large dressing applied to incision with weight of patient against it.

procedure with wound edges well separated by retractors, the wide opening at the site of abscess rupture was plainly visible. With no anesthesia at all it was easy to excise sloughing pieces of lung tissue and to control lung drainage until all nonviable tissue had separated.

Immediately following the wide incision of the parietal pleura, with the introduction of no drains at all, a very large thick dressing previously made ready is applied over the opening even as the pus pours out, and then the patient is promptly placed on his back. The weight of his body plus the immediate saturation of the dressings at once closes the opening so that to all intents and purposes it is airtight, and yet all the pleural fluid is evacuated and does not reaccumulate.

After three or four days the soft tissues tend to occlude the drainage opening. We then insert six or eight one-inch Penrose drains, fastened together with one safety pin. These occasion the patient little discomfort and afford complete and adequate exit for the pus.

Parenthetically, since Penrose drains have proved so valuable in rapid lung reexpansion following ruptured lung abscess, we have tried them in approximately twenty-five cases of comparatively early postpneumonic empyema where the presence of pus between the pleural leaves has not been of sufficiently long standing to cause marked resistance by the visceral pleura to lung reexpansion on evacuation of the empyema. The use of Penrose drains rather than stiff rubber tubes has offered so little resistance to the approximation of the pleural leaves that often within forty-eight hours the lung is out to the chest wall, and breath sounds come through quite distinctly since these soft Penrose drains are squeezed snugly between the parietal and visceral pleura by the reexpanding lung, offering minimal interference to complete lung expansion. When a considerable series of these postpneumonic empyemas have accumulated, they will be reported in detail.

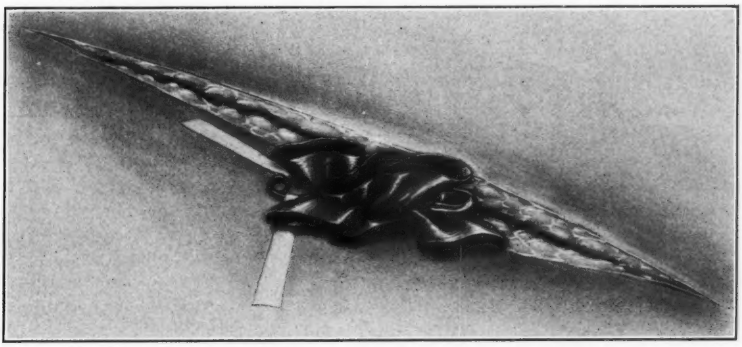


Fig. 4.—Multiple Penrose drains to maintain adequate chest wall opening, to eliminate tube pressure on lung and diaphragm, and to minimize respiratory suction.

In returning to acute suppurative pleuritis following ruptured lung abscess, the following cases are reported.

CASE 1.—E. S., male, 35 years old. There was lung abscess Dec. 22, 1933, posterior to hilum, right lung, as established by vomica and x-ray fluid level. After three days of constant productive cough, sudden extreme pain from the scapular region referred forward around right chest, and there was marked dyspnea. The shock and dyspnea were so great that death seemed near during the first few hours following lung rupture.

A positive diagnosis was made of hydropneumothorax six hours later by aspiration of foul turbid watery fluid. Operation followed immediately. Four-inch sections of ninth and tenth ribs were excised from the border of the erector spinae laterally. The intercostal bundle was excised. The entire lung appeared two-thirds collapsed. In this first case, two thumb-sized black rubber tubes were inserted and placed under water in a bedside bottle. Dry gauze was placed tightly around tubes. A large dressing and adhesive were used in an attempt to seal off the chest wall opening completely except for the rubber tubes mentioned above. By constant supervision and the weight of the patient's body against the dressing, this opening was kept reasonably tight. The greatest difficulty was the presence of the

rubber tubes extending out through the dressing and partially kinked by the patient's body weight. Anoxemia and toxemia were extreme for five days; following this, improvement was rapid and uninterrupted until recovery.

CASE 2.—E. S., male, 24 years old, was sick one month following vomica. X-ray fluid level indicated a lung abscess in the right lower lobe. On Jan. 24, 1936, sudden frightful pain occurred involving the entire lower right half of chest. Profound shock developed rapidly. Four hours after onset, there was external drainage. Five inches of the ninth rib were excised from the transverse process laterally. Pleura was widely opened, and there was tremendous outpouring of pus and inrushing of air. Shock and anoxemia almost overwhelmed the patient at this time. Without insertion of drains, an enormous dressing previously prepared was placed at once over the large pleural opening on the posterior chest wall. The patient immediately turned upon this dressing without taking time to apply binder and was put in the oxygen tent at once.

The improvement in this patient was the most striking of all. In twelve hours breathing was comparatively easy; in forty-eight hours he seemed definitely out of danger. On the fourth day, six one-inch Penrose drains were inserted through the pleural opening. On the fifth day, retraction of wound edges showed a large clean opening representing the roof of the lung abscess. Lung tissue beneath was comparatively clean. No slough was excised. The cough ceased on the eighth day. The patient was discharged from the hospital fifteen days after admission, with lung almost completely reexpanded though bronchopleural fistulas were still audible.

CASE 3.—C. T., female, 24 years old. Vomica, x-ray fluid level proved a lung abscess in the right lower lobe. On the morning of Dec. 19, 1936, rupture took place through the visceral pleura. Excruciating pain occurred over the entire right side, lower half of chest, followed by immediate shock. Patient raised, almost continuously, foul thin watery material. Six hours later, operation was performed in bed. Four-inch sections of the seventh and eighth ribs from the posterior axillary line were removed mesially. Six large Penrose drains, fastened together with a safety pin, were inserted. A very large dressing was immediately applied and, with patient in semirecumbent position, was placed directly against this opening and kept in that position. There was almost no audible sucking through the wound. Improvement was evident at once, and the patient was definitely improved within forty-eight hours. On the seventh day, the wound edges widely separated. A hole in the lung abscess was distinctly seen. Sloughing material was trimmed off with scissors. Coughing ceased and, despite bronchopleural fistulas, the lung almost completely reexpanded on Jan. 10, 1937, twenty-one days following drainage.

Unfortunately, we have but these three cases to report. To wait for a considerable series to accumulate would mean long postponement of this paper. We feel that the results justify early emphasis. The important factors which presumably have favored recovery are: (1) immediate operation; (2) large chest wall opening just lateral to costal vertebral gutter at the most dependent level of accumulated pleural fluid; (3) immediate application of thick voluminous dressing without insertion of drains; (4) placement of patient in semirecumbent position on his back against this dressing in order to make it at once airtight; (5) insertion of several Penrose drains when pleural and soft tissue edges interfere with continuous drainage; (6) postponement of lung visualization and enlargement of abscess opening until patient recovers from initial shock.

REFERENCES

1. Sergent, Emile, and Iselin, Marc: Abscess of the Lung With Pleural Effusions, J. THORACIC SURG. 3: 109, 1933.
2. Neuhof, Harold, and Hirshfeld, Samuel: Putrid Empyema, Ann. Surg. 100: 1105, 1934.
3. Graham, E. A., and Bell, R. D.: Open Pneumothorax: Its Relation to the Treatment of Empyema, Am. J. M. Sc. 156: 839, 1918.
4. Eggers, Carl: Non-tuberculous Pulmonary Suppuration, New York State J. Med. 35: 1068, 1935.

DISCUSSION

DR. H. C. BALLON, Montreal, Canada.—I should like to ask Dr. Dolley if he proposes to employ this operation in the treatment of a pleural effusion complicating putrid lung abscess. I refer to the type of case in which the abscess also constitutes a problem after the effusion is drained. I have in mind too those instances when drainage and not compression of the associated lung abscess is the problem.

DR. HOWARD LILIENTHAL, New York, N. Y.—I was very much interested in this paper. I agree that cases of this sort should be reported without waiting until a larger number have been collected.

I approve absolutely of this method in the cases in which it was used by Dr. Dolley. That it is universally applicable, I am not so sure. It will depend upon the individual patient.

The matter of anesthesia, for example, is very important. I like to do operations with local anesthesia, if possible, especially in children, because there is always the danger in general anesthesia of infecting the opposite lung.

There is one point that Dr. Dolley made in the beginning, and while it has nothing to do with this treatment, it has much to do with the disease itself and its diagnosis. I have spoken of precaution in aspirating in a case of this kind, and of the number of consequent phlegmons of the chest wall which are fatal because they are not discovered until they approach the surface, although they begin in the deeper layers of the cellular part of the chest wall. I have seen many. I would once more advise, therefore, the following method of aspiration whether you think that the collection is putrid or not.

When you have made an aspirating puncture, whether you find fluid or not, the syringe should be disconnected from the needle, still in the suspected tissues, emptied, a little alcohol put into the barrel, reconnected with the needle, the alcohol gently injected into the chest wall during the removal of the needle. There will be no phlegmon following aspiration done in this way.

DR. JOHN V. BOHRER, New York, N. Y.—I wish to mention and recommend the use of zinc peroxide, as advised by Dr. Frank Meleney of New York City. This substance is a non-water-soluble whitish powder and, when applied to a wound, gives off oxygen for a number of hours. It is most efficacious in controlling putrefactive anaerobic infections, such as are often encountered following ill-advised chest aspirations of putrid pus, or following operations for drainage of putrid pus.

I did a lobectomy for a central abscess of the left lower lobe, the urgent symptom having been a repeated and uncontrollable hemorrhage. The patient developed a severe putrefactive infection of the chest wall, which progressed rapidly. The tissue simply "melted away" in spite of the ordinary treatment for such wounds. Zinc peroxide was used with most satisfactory results. I am sure the patient would not have survived if this substance had not been available.

Zinc peroxide may be used inside the pleural cavity, but it is sometimes removed with difficulty. I can recommend this substance as a great addition to our armamentarium for the control of these chest wound phlegmons.

DR. FRANK S. DOLLEY.—I am glad that Dr. Ballon brought up the question concerning the care of the ruptured lung abscess itself at the time of the initial operation for pleural drainage. It gives me an opportunity to re-emphasize the vital importance to the patient of minimizing the extent of the surgery at this crucial moment. It is my conviction that a considerable percentage of the European fatalities has been largely due to the fact that the surgeons have tried to unroof the lung abscess at the time of the drainage of the empyema. These patients are already appallingly anoxemic. The sudden wide opening made in the chest wall precipitates under the most favorable conditions a fearful decrease in the amount of oxygen available to the patient even with the most extreme inspiratory effort. This external opening must be blocked at the earliest possible moment. Not minutes, but seconds, are valuable. Using retractors to expose the lung widely and spending several minutes in identifying and trimming off necrotic lung tissue, I believe, not only is unwarranted at this time but also is probably the deciding incident in the fate of the patient. It is infinitely safer to wait one or more days before attempting to deal with the abscess itself.

Dr. Lilienthal spoke of the anesthesia for these desperately sick people. We use only a local, 1 per cent novocaine; and often transfer the bed itself to the operating room in order to minimize any effort that would be required on the part of the patient in moving from the bed to the stretcher and thence to the table. Even this small amount of increased exertion might demand more oxygen than there is to spare. In one case we operated with the head of the patient in an oxygen tent. If operation is not immediately to follow diagnostic aspiration of the pleural fluid, we inject iodine along the tract as we remove the needle through the soft tissues. We have found iodine to be as efficacious as any other fluid in the prevention of cellulitis following this procedure and the least irritating of any we have tried.

Dr. Bohrer's discussion concerning cellulitis of the chest wall is an important one. Advancing cellulitis, secondary to needle aspiration in the presence of putrid empyema, if not controlled immediately is almost invariably fatal. It is our practice if operation for one reason or another cannot be promptly performed, to have an intern or nurse check every hour for beginning cellulitis. When it is evident that infection is starting to spread beneath the deep fascia, operation is at once imperative. In a comparatively small area, incision is carried well through this area of soft tissue inflammation, the edges are undermined and held apart by Penrose drains. If it has become extensive, however, we believe that enormously long incisions with elevation of the flaps by strong blunt dissection cause so much damage to those barriers which nature is raising against the spread of this inflammation that this surgical procedure itself and the subsequent increased absorption that follows it rapidly overwhelm the patient.

There was no cellulitis of the chest in the cases described in this paper. The patients were operated on immediately following aspiration. But in the other types of empyema where cellulitis has developed, we have attempted to minimize the surgical trauma incident upon wide opening of these areas of cellulitis by making a series of transverse incisions, one at the most dependent part of the cellulitis, and others, as indicated, several inches above and parallel to this lowest one. Penrose drains then are very gently slipped from below upward, a matter only of two or three inches, in the attempt to completely separate the roof from the floor in the infected area. Usually this halts the advancing process. Injury of lymph and blood vessels is minimized.

PRIMARY CARCINOMA OF THE TRACHEA*

ORMOND S. CULP, M.D.
MONTREAL, CANADA

PRIMARY tumors of the trachea constitute a comparatively rare group of lesions that are of particular clinical and pathologic interest. The clinical aspect is concerned with methods for making a correct early diagnosis and instituting more effective treatment, while the pathologic problem centers about the morphology of the tumor and its possible malignant tendency. This brief report is confined to the pathology of carcinoma of the trachea. An attempt will be made (1) to review the literature, (2) to make an analysis of the types of primary carcinoma that already have been described, (3) to record the anatomic findings in two additional cases.

As early as 1767 Lieutaud[9] described a fibroma in the trachea as an incidental autopsy finding. No other tumors were described until after the introduction of the laryngoscopic mirror in 1854. Rokitansky[104] reported an autopsied case in 1857 which was one of the first malignant tumors to be observed. Türck[16] in 1861 first utilized the newly discovered indirect laryngoscopy to diagnose a tracheal growth in the living subject, but Langhans[79] in 1871 was the first to report a primary carcinoma confirmed by detailed histologic study.

The development of the bronchoscope did much to aid the early and accurate diagnosis of these tumors. Killian[19] first diagnosed a primary tumor of the trachea by endoscopy. Reports of tracheal growths became progressively more frequent with improved laryngoscopic and bronchoscopic methods and with more complete postmortem studies.

INCIDENCE

In 1898 von Bruns[124] assembled the available statistics on primary tumors, in general, after having observed 7 such cases. These were classified as shown in Table I. They totaled 147 cases, of which 21 per cent were carcinomata. Krieg[7] made the next comprehensive study in 1908 and found a total of 201 cases with no appreciable change in the proportion of carcinoma. Fifty-one additional newgrowths were collected by Lombard and Baldenweck[10] in 1914, while in 1929 D'Aunoy and Zoeller[41] presented the last exhaustive survey of the subject with a total of 351 cases. These included 91 instances of carcinoma (see Table I).

*From the Pathological Institute, McGill University. Professor Horst Oertel, Director.
Received for publication, May 29, 1937.

An attempt has been made to complete these general statistics by including all the reported primary growths to the end of June, 1936. The classification used in the previous studies has been followed as closely as possible. Most of the literature during the past seven years has dealt with malignant tumors. Of the 82 new cases of primary growths recorded during this period, 56 were described as carcinomata. Of the total 433 primary tumors on record at the end of June, 1936, 147 (or 34 per cent) were carcinomata (see Table I).

TABLE I

PRIMARY TRACHEAL TUMORS

TYPE	VON BRUNS 1898	KRIEG 1908	LOMBARD & BALDENWECK 1914	D'AUNOY & ZOELLER 1929	PRESENT STUDY 1936
Fibroma	23	25	29	33	36
Papilloma	33	41	51	59	63
Lipoma	3	4	5	5	5
Chondroma, osteoma, tracheopathia osteoplastica	29	42	47	65	71
Adenoma	5	6	6	8	9
Lymphoma	2	2	2	3	4
Intratracheal goiter	7	14	19	25	28
Amyloid tumors	–	–	1	2	2
Mixed tumors	–	–	–	2	3
Angioma	–	–	1	2	2
Cylindroma	–	–	–	1	1
Sarcoma	14	21	23	26	31
Carcinoma	31	40	54	91	147
Carcinosarcoma	–	–	–	1	1
Endothelioma	–	–	–	1	1
Histology undetermined or doubtful	–	6	14	27	29
Total	147	201	252	351	433

Although primary carcinoma appears to be the most frequent type of malignant tumor of the trachea, the following observations indicate that it is of relatively rare occurrence. In 5,063 autopsies Fraenkel[50] found 7 cases. This is the highest percentage (1.4) of any series on record. From 1918 to 1930, Figi[48] reported 5 patients with primary carcinoma at the Mayo Clinic. D'Aunoy and Zoeller[41] were able to find only one case at the Charity Hospital of New Orleans over a period of twenty-three years. They point out that statistics from the Pathological Institute of Basle for thirty-five years showed only one growth of tracheal origin out of 1,078 cases of malignancy. Broman[34] added one case from a series of 545 malignant growths found at autopsy at the Cook County Hospital, Chicago. Bilz[2] found none of tracheal origin among 700 cases of carcinoma at the University of Jena. Bejachs[1] was unable to find any among 1,300 cancer autopsies at the Berlin Charité, and Mielecki[12] was also unsuccessful in his review of 560 cancer patients. Stenn[114] contributed the only case at the Billings Hospital, Chicago, and cited Lund as having found 2 cases in 25,000 autopsies

at the Philadelphia General Hospital from 1867 to 1934. He also noted that Symmers found none at the Bellevue Hospital, New York, that Evans found none in 12,000 autopsies at the Los Angeles County Hospital, and that Wright found none in 5,000 autopsies at Guy's Hospital, London.

A personal survey yielded no primary carcinoma of the trachea in 15,000 autopsies at the Johns Hopkins Hospital, Baltimore, none in 7,000 autopsies at the Baltimore City Hospitals and none in 3,900 autopsies at the New Haven Hospital. The cases herein reported are (1) the only carcinoma of tracheal origin in 9,000 autopsies at the Pathological Institute of McGill University, Montreal, and (2) the only one in 12,700 autopsies at the Montreal General Hospital.

CLINICAL ANALYSIS

There is very little about the clinical picture of the disease which is characteristic. A multitude of initial symptoms has been described for the various cases in this series, but none are indicative of carcinoma per se. The commonest early symptom was a tickling sensation in the trachea, followed by an irritating cough and persistent hoarseness (due to pressure on the recurrent laryngeal nerve). In some instances, however, this entire phase was lacking, and the earliest symptom was dyspnea or hemoptysis.

According to Jackson[4] the chief symptoms of any tracheal tumor are: (a) wheezing respiration, (b) nocturnal attacks of dyspnea, (c) dyspnea on exertion, (d) asphyxia, unless averted. Gilfoy[52] claimed that the symptom complex of intermittent dyspnea, cough, perhaps occasional hemoptysis, apparent good health and absence of obvious intrathoracic signs suggests a tumor in the tracheobronchial tree and warrents endoscopy. Because of the mildness of early symptoms, most patients failed to seek medical attention until the process was advanced. The chief diagnostic distinctions were asthma, the more common mediastinal tumors, carcinoma of the esophagus, empyema, bronchiectasis, pulmonary abscess, pneumonia, and enlarged thyroid. All writers agree that diagnosis depends upon the combined methods of palpation, x-ray, laryngoscopy, bronchoscopy, esophagoscopy, and occasionally open exploration.

Almost every conceivable form of therapy has been tried, varying from the more simple endoscopic measures to radical surgery. Figi[48] offered the following as a summary of the treatment of tracheal carcinoma: (a) Lesions in the upper half are best treated surgically with exposure of the growth by tracheofissure and destruction of it with electrocoagulation. A large tracheal tube is then worn from six months to one year and simplifies later treatment if necessary. (b) Excision of a segment of trachea has been carried out in some cases but seems

to offer no better results than diathermy and involves greater risks. (c) Implanted radium and surgical diathermy are recommended for the lower half of the trachea. (d) In all cases the regional glands should be given radium or deep x-ray therapy.

Regardless of the treatment, the prognosis for carcinoma of the trachea is generally very poor. Apparently the only real hopes for cures are the early lesions in the upper half, and even then the disease is not recognized usually until well advanced. Most patients in this series died within one year, but a few isolated patients were living as long as seven years after the initial treatment.

The terminal event was usually (1) suffocation, (2) pneumonia, frequently in instances of tracheoesophageal fistulas, (3) massive hemorrhages.

PATHOLOGIC ANALYSIS

Data concerning carcinoma are frequently misleading because accurate clinical diagnoses are seldom made, histologic reports are meager and postmortem confirmation is often lacking. It is possible that some cases in this review should not have been included, while other cases of mediastinal tumors of uncertain origin should not have been omitted. Such errors of commission and of omission probably would about balance each other. This analysis is based, therefore, on a review of the 147 recorded cases.

A. *Age Distribution.*—The youngest patient on record was a girl 18 years of age, while the oldest patient was an 82-year-old man. Of the 111 reports in which the patient's age was given, 100 cases (90 per cent) were distributed fairly equally among persons between 30 and 70 years of age with only slightly increased frequency in the fifth and sixth decades (see Table II).

B. *Sex Distribution.*—Practically every reviewer has emphasized the marked predominance of males as victims of tracheal cancer. Sex was recorded in 116 cases of the present series and, of these, 63 per cent occurred in men (see Table II).

TABLE II

AGE	SEX			METASTASES			TOTAL CASES
	MALE	FEMALE	NOT GIVEN	PRESENT	NONE	NOT GIVEN	
10-19	0	1	0	0	1	0	1
20-29	4	1	0	1	3	1	5
30-39	11	9	0	9	8	3	20
40-49	14	14	0	14	8	6	28
50-59	21	10	0	18	6	7	31
60-69	15	6	0	14	3	4	21
70-79	3	1	0	3	0	1	4
80-89	1	0	0	1	0	0	1
Not given	4	1	31	2	0	34	36
Total	73	43	31	62	29	56	147

C. *Location of Tumor.*—The trachea has been divided arbitrarily into the upper, middle, and lower thirds. Carcinoma occurred most frequently near the bifurcation, i.e., in the lower third. Next in frequency was the upper third, and least often it was situated in the middle portion (see Table III).

TABLE III

	ANNULAR	ANTERIOR WALL	ANTERIOR AND LATERAL	POSTERIOR WALL	POSTERIOR AND LATERAL	LATERAL ONLY	NOT GIVEN	TOTAL CASES
Upper third	3	3	5	11	9	4	10	45
Middle third	0	2	0	5	2	1	10	20
Lower third	2	10	4	16	3	4	22	61
Not given	0	1	1	3	0	0	16	21
Total	5	16	10	35	14	9	58	147

The tumor formed an annular mass about the trachea in only five instances. It was usually a well-localized process in the wall similar to the first case herewith reported (see Fig. 1). By far the commonest area for involvement was the posterior wall alone, and when combined with those involving both the posterior and lateral walls, these tumors constituted 55 per cent of all the cases in which an accurate location was given (see Table III).

According to this study, therefore, the commonest site of carcinoma of the trachea is in the posterior wall of its lower third.

Strauss[14] expressed the belief that the frequent involvement of the posterior wall is due to the drainage of lymph posteriorly. Von Bruns[124] and Schrötter[109] attributed it to the predominance of glandular epithelium in the posterior wall, in contrast to the cartilaginous nature of the anterior wall. On the same basis they explained the infrequency of circumferential growths. Simmel[111] claimed that the lower third of the trachea is most exposed to trauma and upon this basis explained the frequency of tumor growths in this region.

D. *Metastases.*—Of the 147 cases reviewed, metastases were mentioned as being present or absent in only 91 instances. In 62 cases (68 per cent) metastases were described, while they were definitely absent in only 29 cases (see Table II). There seems to be no correlation between the incidence of metastases and age periods. Thus the decades in which carcinoma is most frequent show a corresponding increased incidence of metastases.

It is a peculiar feature that the commonest type of carcinoma to metastasize was the squamous cell variety. Of the 38 cases recorded, 23 had definite metastases (see Table IV).

The site of the secondary growths varied greatly, but they were usually regional, involving the esophagus (other than by direct extension), the mediastinal, peribronchial and cervical glands, and occasionally the lungs. Isolated instances of metastases to the skeleton, gastrointestinal tract, liver, spleen, pancreas and kidneys were recorded also. As D'Aunoy and Zoeller[41] pointed out, these tumors are in contradistinction to the laryngeal carcinomata which rarely metastasize and show a predilection for a certain limited number of organs. It appears that primary carcinoma of the trachea may be associated with secondary growths in any tissue or organ.

E. Histogenesis.—According to Maximow,[11] the epithelium of the trachea is of the ciliated, pseudostratified, columnar type and rests on a distinct basement membrane. Numerous goblet cells are scattered throughout this epithelium, and the lamina propria contains many small glands like those of the larynx. These glands are mostly external to the elastic fibers and open by short ducts on the free epithelial surface. With this histologic structure in mind, one would expect the adenoma and adenocarcinoma cell to be a frequent constituent of tumors of the trachea. Thus Langhans[79] in 1871 made the first important histologic study of tracheal carcinomata and concluded that most of them were derived from the epithelial cells of the mucous glands. This view was supported by the later investigations of Virchow[122] and Hamacher.[56]

TABLE IV

DIAGNOSIS	METASTASES	NONE	NOT GIVEN	TOTAL CASES
Definite Morphology:				
Squamous-cell carcinoma	23	5	10	38
Adenocarcinoma	10	6	10	26
Basal-cell carcinoma	1	5	2	8
Cylindric-cell carcinoma	3	2	2	7
Columnar-cell carcinoma	0	1	2	3
Uncertain Morphology:				
Alveolar carcinoma	2	2	2	6
Papillary carcinoma	1	1	2	4
Medullary carcinoma	10	0	3	13
Scirrhus carcinoma	2	0	1	3
Unclassified:				
Carcinoma	8	6	19	33
Epithelioma	2	1	3	6
Total	62	29	56	147

Considerable confusion then resulted from the contradictory statements of later authors concerning the predominant cell types in these tumors. Oppikofer[95] considered the squamous-cell carcinoma to be the most common, while Fraenkel[50] openly disagreed. Kaufmann,[5] quoting Nager, Oestereich, Schmiegelow and Kahler, thought the squamous cell variety was the one most frequently encountered, while Figi[48] contended

that adenocarcinoma was the most common. Hence the literature offers a host of conflicting statements which have not been clarified in the more recent pathologic text and reference books.

A histologic survey of the 147 cases was attempted, but, because of differences in terminology and the lack of adequate microscopic data, many cases were very difficult to classify accurately. Furthermore, earlier observers followed a very indefinite classification of cancers. Some were diagnosed on a morphologic basis (which is the only acceptable method), while other authors described only the general tumor cell arrangement and were content to identify the growths merely as carcinoma or epithelioma (see Table IV).

In general, the carcinomata seem to fall into two main groups, those arising from (1) glandular epithelium and (2) surface epithelium. The predominant cell in the latter group may be columnar, cylindric, basal, or squamous. Of these, the squamous-cell carcinoma is the most common. Thirty-eight unquestionable cases were found in this series. Only 26 definite adenocarcinomata were found, but it is probable that many of the alveolar and medullary growths belong in this group (see Table IV). It is noteworthy that the adenocarcinoma does not predominate as the early writers believed, but it would appear that the squamous-cell carcinoma is equally as common.

It is not difficult to explain the occurrence of the adenocarcinoma. It arises from the mucous glands of the trachea. The presence of mucoid secretion is noteworthy evidence. Inasmuch as no squamous epithelium is found in the normal trachea, the occurrence of squamous-cell tumors presents a morphologic problem. Two schools of thought have evolved, each supported by histologic evidence.

(1) Drasch in the Anatomie Humaine (Poireo) pointed out that islets of squamous epithelium frequently occur in the trachea as embryonic arrests. Such epithelial islands have been observed repeatedly by many investigators in the absence of neoplastic diseases. Inasmuch as both the trachea and the esophagus develop from the embryonic fore-gut, it is thought that nests of esophageal epithelium are included in the tissues of the trachea at the time it separates from the ventral portion of the esophagus. Reiche,[102] Nager,[89] Deland and McFarland,[44] Heymann,[62] and a few others were convinced that the tumors of their experience were best explained on the basis of Cohnheim's theory of embryonal inclusions. In the absence of any evidence of cellular metaplasia, this explanation seems quite justified.

(2) Many tumors, however, showed unmistakable evidence of metaplasia. Breslich[32] was able to follow the steplike transition from columnar ciliated epithelium to squamous cells. The tumors reported by von Meyenburg,[125] Gilfoy,[52] Baratoux,[19] Nielson,[91] Barth,[22] Teubert,[117] and Tiling[119] also illustrated varying degrees of metaplasia.

Krompecher[8] studied basal- and squamous-cell carcinomata which occurred in structures lined by columnar epithelium and decided that these tumors originated from proliferation of the basal cells. He claimed that these undifferentiated cells are morphologically similar to the basal cells of stratified epithelium and are distributed irregularly on the basement membrane of the mucosa between the cylindric cells. Normally they divide and differentiate into columnar epithelium to replace the lining cells which are injured or desquamated, but under certain conditions they may differentiate into transitional or stratified squamous epithelium and give rise to basal- or squamous-cell carcinomata.

In the trachea of rabbits with acute inflammations, Kawamura[6] observed a metaplasia of columnar ciliated to transitional epithelium. Teutschlaender[15] noticed that the bronchi of certain rats after bronchopneumonia were lined by transitional or stratified squamous or even hornified squamous epithelium, and traced the development of these changes step by step from the basal cells.

Goldzieher[3] described parts of the bronchi of a patient who had died of diphtheria as being lined by a wide layer of basal epithelium. In different places this was covered by either cylindric or squamous cells. In his opinion these unusual changes resulted from a further differentiation of the proliferating basal cells to cylindric or squamous types.

Three squamous-cell carcinomata of the lung described by Siegmund[13] developed in bronchiectatic cavities. The single-layered, cylindric, epithelial lining of the dilated bronchi was transformed into a many-layered, basal or transitional epithelium from which Siegmund thought the tumors originated. There can be no doubt, therefore, that metaplasia *does* occur in the epithelium of the air passages, and it seems probable that this frequently precedes neoplasia. Case 1 adds further support to this view.

CASE REPORTS

CASE 1.—*Clinical Course:* This 68-year-old man had increasing dyspnea and dysphagia for six months before coming to the hospital. Physical examination revealed marked respiratory stridor with indrawing of the intercostal spaces, emaciation, complete paralysis of the left vocal cord, slight left facial paresis and a firm nodule the size of a marble between the origins of the left sternocleidomastoid muscle; x-ray pictures showed displacement of the trachea and mediastinum to the right by a solitary dense shadow at the level of the third rib. Both lung bases were congested and there was a moderate polymorphonuclear leucocytosis. The clinical impression was a mediastinal tumor with lobular pneumonia.

Because of the pneumonia, no instrumentation was attempted. The patient had a fever of 99° to 102° for the next three weeks with periods of temporary improvement following repeated administration of morphine and atropine. For several days he was able to take a semisolid diet fairly easily. He became progressively weaker, however, and had episodes of severe chest pain, persistent coughing and a low grade fever. He died forty-three days after admission to the hospital with the clinical picture of diffuse bronchopneumonia complicating a mediastinal tumor of unknown origin.

Postmortem Findings: (A-386-36) Exposure of the thorax showed the upper mediastinum to be the site of an extensive tumor mass. The trachea was displaced 1 cm. to the right of the midline by the tumor, and its lumen seemed to be narrowed to about half its normal size.

On opening the larynx and trachea there was a firm, gray mass on the left lateral wall 5 cm. below the vocal cords. This was 4.8 cm. long and 2.5 cm. wide. The tumor involved all coats of the trachea producing a firm, granular, ulcerated surface 3 cm. long and 1.6 cm. wide. The mass reached a level 2.4 cm. above the left main bronchus (see Fig. 1). The transverse sections showed that the tumor extensively involved the left mediastinal tissues, forming an irregular, firm, gray mass 4.4 cm. wide. This extended posteriorly 5.6 cm. where it became firmly attached to the thoracic vertebrae (see inset Fig. 1).

The left subclavian artery formed its left lateral border and was partly surrounded by the mass. The innominate artery extended up the right lateral border. The left common carotid artery was surrounded completely by tumor and crossed to lie beside the left subclavian. The left innominate vein was filled with a firm, gray thrombus resembling tumor tissue. The left internal jugular vein was dilated to 1.2 cm. in diameter and was filled with laminated, pale and dark red clot. The other great vessels in the neck and mediastinum were free.

Inferiorly the tumor extended to the aortic arch and superiorly reached the level of the isthmus of the thyroid, the left lower pole of which was adherent to the capsule. The supraclavicular lymph glands were dissected and found to be free of tumor.

The esophagus was displaced to the left and formed the lateral border of the tumor in its lower half. The coats were invaded, producing a firm, gray, oval, nodular mass 3 cm. long and 2.8 cm. wide, over which the mucosa was intact except for an area 6 mm. in diameter near its center (see Fig. 1). This showed a shallow defect, the floor of which was flat and pale gray. The esophageal lumen was narrowed to about half its normal diameter by the bulging tumor mass.

On the posterior surface of the esophagus, 3.5 cm. below the lower limit of the tumor, there was a small, gray, granular elevation 1 cm. long and 6 mm. wide. On section this appeared as an irregular thickening of the mucosa, while other coats were not definitely indurated. There was a similar, slightly smaller, granular area 3.8 cm. below this which also showed no evidence of induration (see Fig. 1). These two areas resembled metastatic growths.

The right pleural cavity contained 175 c.c. of cloudy yellow fluid, and there were numerous loose pleural adhesions. There were also firm adhesions at the base of the left pleura. Both lungs showed the gross and microscopic picture of a diffuse aspiration bronchitis and purulent bronchopneumonia.

Incidental findings included multiple gastric ulcers, glandular hyperplasia of the prostate, multiple false diverticula of the sigmoid, productive cholecystitis with cholelithiasis, focal arteriosclerotic gliosis in the right cerebrum, patchy pachymeningitis hemorrhagica, and cloudy swelling of the parenchymatous organs.

Tumor Histology: The upper two-thirds of the tumor consists of cells in loose alveolar arrangement. These resemble transitional epithelium. They have large, pale, round, or oval nuclei with scattered nucleoli. There are many degenerating forms and the larger cell masses show central necrosis. Between these cells are a few, scattered, hyperchromatic, irregular, spindle-shaped nuclei without fibrils. In several places the apparently transitional cell replaces the marginal columnar epithelium of the trachea where the basement membrane is completely lost (see Fig. 4).

Sections across the lower third of the tumor show masses of immature pavement epithelium with typical cancer whorls and abundant keratinization, replacing

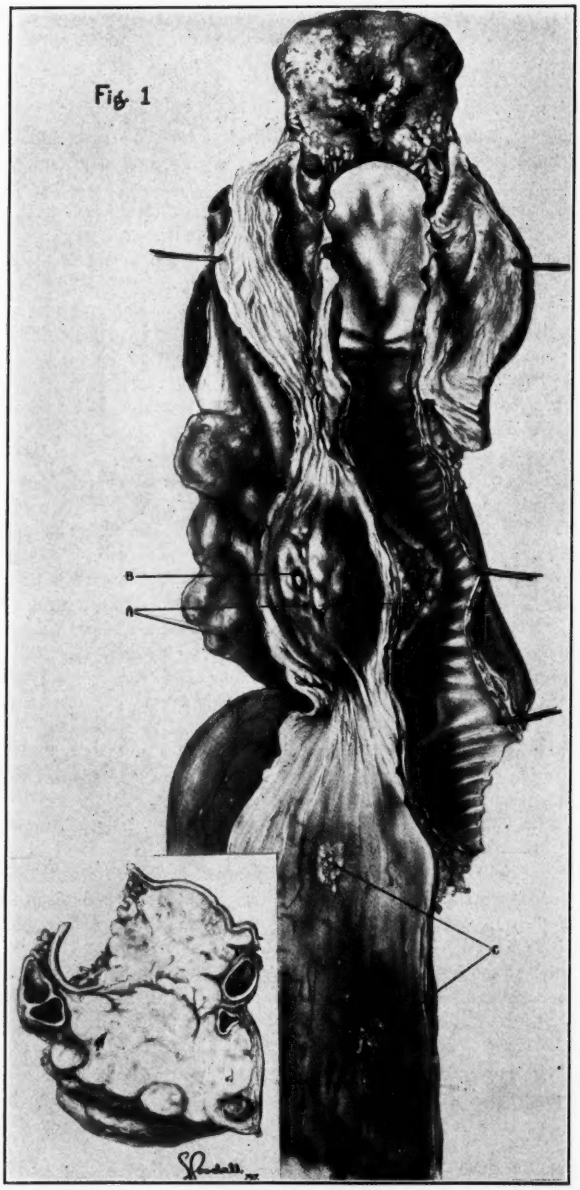


Fig. 1.—Posterior view of the opened trachea and esophagus. *A*, The tumor mass displaces the trachea to the left, all coats are infiltrated and there is extensive ulceration. *B*, Small erosion of the esophageal mucosa over the bulging tumor. *C*, Metastases in the lower esophagus.

Inset represents a cross section at the level *A*. The tumor replaces the left tracheal wall and fills the mediastinum. The intact overlying esophageal mucosa is seen in the upper right, and the thrombosed left innominate vein is at the lower right border.

the tracheal coats and surrounding cartilaginous rings (see Fig. 2). These cells show no evidence of intracellular bridges. There is here an abundant, dense, fibrous tissue stroma. The immature squamous epithelium in places fuses with the immature transitional cells described above (see Fig. 3). The tumor cells

Fig. 2.—Low power photomicrograph of a transverse section from the lower third of the tumor. The trachea above may be identified by the cartilaginous ring. The wall of the trachea is infiltrated with immature keratinizing squamous epithelium. The esophagus below is partly invaded by a solid mass of transitional epithelium. In the center of the picture is a zone of metaplasia. (×50).

invade many mucous glands, but there is no evidence of mucoid secreting neoplastic forms. Many lymphatics are dilated and filled with both immature squamous and transitional cells.

The esophageal epithelium is hyperplastic with intact basement membrane over the tumor, except for the small defect noted grossly. At this area there is a narrow zone of necrosis and underlying fibrous tissue. There is no evidence of neoplastic cell invasion at the margins of this defect. The granular elevations in the lower part of the esophagus (Fig. 1) show replacement of the hyperplastic epithelium by masses of immature squamous cells (see Fig. 5). These cells lie mostly in dilated lymph spaces and are confined to the mucosa and submucosa.

Fig. 3.—High power photomicrograph of Fig. 2 at the junction of the squamous and transitional cells. Note the keratinization at the extreme right. The adjoining column of immature squamous cells gradually fuses with the more loosely arranged surrounding transitional cells. (×280.)

Fig. 4.—Photomicrograph of the trachea from the upper third of the tumor. A wedgeshaped mass of dark staining carcinoma cells replaces the marginal columnar epithelium and extends downward radially from the mucosa. (×280.)

Anatomical Summary: Metaplastic squamous-cell carcinoma of the trachea. Direct invasion of the mediastinum, regional lymph glands, and left innominate vein. Lymphogenous metastases to the esophagus. Diffuse aspiration bronchopneumonia.

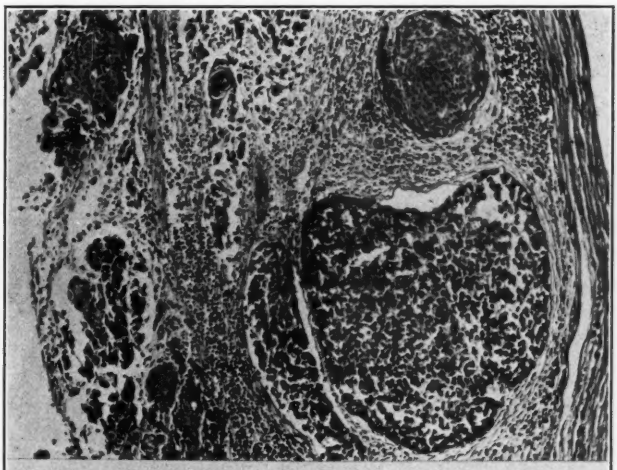


Fig 5

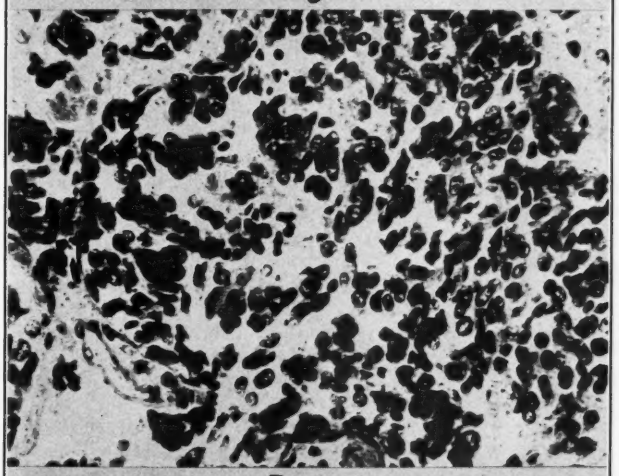


Fig 6

Fig. 5.—Photomicrograph of one of the esophageal metastases showing the irregular mucosa containing masses of immature squamous cells. Three lymphatic channels are dilated and plugged with tumor cells. The stretched and intact muscular coat is at the extreme right. (×240.)

Fig. 6.—High power photomicrograph of the main tumor mass in Case 2 showing immature squamous epithelial cells and smaller hyperchromatic irregular columnar cells. (×580.)

CASE 2.—*Clinical Course:* This patient was a 54-year-old woman whose illness began May, 1908, when she swallowed a fish bone which stuck in her throat and

was extracted later at home. During the same month she first noticed the sensation of a marble moving up and down in her throat. This became gradually worse with increasing dysphagia. During the next nine months dyspnea was slowly progressive until inspiration became forced and almost gasping. A small round hard slightly tender lump appeared on the right side of the neck near the thyroid cartilage in August of the same year. This increased in size until November and then remained essentially the same size until her admission three months later.

Physical examination showed emaciation, intense dyspnea and orthopnea, some respiratory stridor, marked dysphagia, enlargement of both lobes of the thyroid with fixation to the surrounding tissues, and signs of emphysema. The clinical impression was malignant goiter.

She had repeated episodes of severe coughing and cyanosis. On the day following admission she was said to have been unable to breathe for about a minute, became black in the face and responded finally to strychnine. Two days after admission the thyroid was exposed under general anesthesia and part of the right lobe resected. As this procedure was completed, the patient suddenly stopped breathing and a tracheotomy was done at the level of the third, fourth and fifth tracheal rings. It was difficult, however, to insert the cannula because of infiltration of the wall, and by the time the emergency measure was completed the patient failed to respond to any stimuli and died apparently of asphyxia.

Postmortem Findings: (M. G. H., A-24-09) Autopsy revealed absence of the right lobe of the thyroid and the tracheotomy wound at the level described above. The latter opened into the middle of a large tumor mass which accounted for the difficulty in passing the tracheal cannula. The lumen barely admitted a No. 10 English rubber catheter. When the larynx and trachea were opened, the vocal cords appeared normal, but a tumor was found to extend from the lower end of the thyroid cartilage downward 4 cm. on the left side of the trachea and then slant off toward the right for an additional 3 cm. At one point it completely encircled the trachea, was 4 cm. wide and 2 cm. in its greatest anteroposterior dimension. The mass also extended into the region of the right lobe of the thyroid where the recent operative procedure had severed part of the tumor. There was some ulceration of the tracheal mucosa below the cricoid cartilage on the left posterior wall, and the tumor presented a very firm, grayish-white surface which cut with much resistance.

The esophageal lumen was so narrowed that it admitted a small penholder with difficulty. This narrowing was due to the pressure of the tumor, described above, encroaching on the lumen of the esophagus from the front. The mucosa appeared everywhere intact.

The lungs showed emphysema. Other incidental findings included productive pleurisy and peritonitis, brown atrophy of the myocardium, passive congestion of the liver and slight arteriosclerosis.

Tumor Histology: Sections show irregular masses of large cells with hyperchromatic nuclei in which nucleoli are numerous. Between these forms are diffusely scattered columnar-shaped cells. These tumor cells are divided into irregular masses by bands of dense fibrous tissue. Mitotic figures are numerous. There is no evidence of keratinization or whorl arrangement (see Fig. 6).

Anatomical Summary: Metaplastic transitional-cell carcinoma of the trachea with invasion of the mediastinum and thyroid.

SUMMARY

(1) Of the 433 primary tumors of the trachea on record at the end of June, 1936, 147 or 34 per cent were carcinomata.

(2) The clinical symptoms were essentially the same as those for any tracheal neoplasm and most of the patients died within one year of the initial examination.

(3) The highest incidence of carcinoma was in males between 40 and 60 years of age.

(4) The tumors were located most frequently in the lower third of the trachea on its posterolateral walls.

(5) Metastases were found in 68 per cent of the cases and were mostly regional.

(6) Adenocarcinoma and squamous-cell carcinoma were the commonest types and were found in about equal frequency.

(7) The adenocarcinomata probably originate in the mucous glands of the trachea.

(8) Some of the squamous-cell tumors were assumed to be newgrowths from embryonic arrests of esophageal epithelium in the trachea, while others were assumed to be the result of metaplasia.

(9) Two case studies of primary carcinoma of the trachea have been added to this series.

NOTE: The first case report was obtained from the records of the Montreal Homeopathic Hospital (No. 1743-36) and the second one from the files of the Montreal General Hospital (No. 172-S-09). I am deeply grateful to the staffs of both institutions for their cooperation. I especially wish to acknowledge my indebtedness to Dr. W. H. Chase of the McGill Pathological Institute for his valuable assistance in preparing the pathologic reports of these cases, and to Miss Shirley Goodall for her excellent illustration.

REFERENCES

1. Bejachs: Ztschr. f. Krebsforsch. **16**: 159, 1917.
2. Bilz: Ztschr. f. Krebsforsch. **19**: 282, 1923.
3. Goldzieher: Centralbl. f. allg. Path. u. path. Anat. **29**: 506, 1918.
4. Jackson: South. Surgeon **5**: 256, 1936.
5. Kaufmann: Lehrbuch der Pathologischen Anatomie, Vol. I, p. 335, 1922.
6. Kawamura: Virchows Arch. f. path. Anat. **203**: 420, 1911.
7. Krieg: Beitr. z. klin. Chir. **58**: 162, 1908.
8. Krompecher: Beitr. z. path. Anat. u. z. allg. Path. **72**: 163, 1924.
9. Lieutaud: Historia an. med., 1767, Lib. IV, Obs. 64.
10. Lombard and Baldenweck: Ann. d. mal. de l'oreille, du larynx **40**: 491, 1914.
11. Maximow: A Text-Book of Histology, 1930, W. B. Saunders Company, Philadelphia, p. 439.
12. Mielecki: Ztschr. f. Krebsforsch. **13**: 505, 1913.
13. Siegmund: Virchows Arch. f. path. Anat. **236**: 191, 1922.
14. Strauss: Ann. Otol. Rhin. & Laryng. **31**: 715, 1922.
15. Teutschlaender: Centralbl. f. allg. Path. u. path. Anat. **30**: 443, 1919.
16. Türck: Klin. d. Krankh. d. Kehlkopfes. p. 502, 1886.

CARCINOMA CASE REPORTS (147)

17. Adam: J. Laryng. & Otol. **30**: 64, 1915.
18. Ascherborn: Arch. f. klin. Chir. **25**: 162, 1880.
19. Baratoux: Ann. d'oto-laryng., 1933, p. 1272 (2 cases).
20. Bargum: Inaug. Diss., Kiel, 1897.
21. Barmwater: Hospitalstid. **73**: 44, 1930.
22. Barth: Ztschr. f. Hals-, Nasen-u. Ohrenh. **35**: 194, 1934.
23. Beebz: Rev. mens. de laryng. 11, 1885.
24. Berens: Tr. Am. Laryngol. Assoc. N. Y., p. 70, 1909.
25. Berger: Bull. Assoc. franç. p. l'étude du cancer **2**: 9,629, 1922.

26. Billroth: Chir. klin. Wien. Berlin, 1879.
27. Bircher: Arch. f. Laryngol. u. Rhinol. **20:** 443, 1908.
28. Blix-Kjellberg: Hygiea, 1872, Jahresb. v. Virchow-Hirsch. **2:** 123, 1873.
29. Bjorlin and Breggren: Schwedische Artzegesellschaft, Sektion für Otrarie, Rhin. u. Laryng., Stockholm, Hygiea **87:** 12, 481, 1925.
30. Borries: Hospitalstid. **68:** 83, 1925.
31. Boschi: Bull. d. sc. med., Bologna **11:** 314, 1900.
32. Breslich: J. Cancer Research **14:** 144, 1930.
33. Broechaert: See Lombard and Baldenweck (Ref. 10).
34. Broman: J. Cancer Research **8:** 394, 1924.
35. Calamida: Sanderdruck aud otti d. con. a. aos. ital. di laryng. otol. e. rhinol., Perugia **10:** 2, 1922.
36. Cayce: South. M. J. **14:** 422, 1921.
37. Chevallier: Lyon méd. **146:** 309, 1930.
38. Chrenov: Mosk. Otolog. Rhinol. Laryng. Gesellsch. **10:** 3, 1928.
39. Claus: Med. Klin. **20:** 567, 1924.
40. Clavel and Mounier-Kuhn: Lyon méd. **17:** 1932.
41. D'Aunoy and Zoeller: Arch. Path. **11:** 589, 1931.
42. Davis: Radiology **5:** 342, 1925.
43. Delafield: New York State J. Med. **36:** 406, 1882.
44. Deland and McFarland: J. A. M. A. **43:** 647, 1904.
45. Dumas and Guichard: Lyon méd. **48:** 1931.
46. Ehrlich: Monatschr. f. Ohrenh. **30:** 121, 1896.
47. Feuchtinger: Monatschr. f. Ohrenh. **61:** 182, 1927.
48. Figi: Arch. Otolaryng. **12:** 446, 1930 (5 cases).
49. Fischer: Monatschr. f. Ohrenh. **14:** 12, 1882.
50. Fraenkel: Deutsches Arch. f. klin. Med. **135:** 184, 1921 (8 cases).
51. Gaillard: Congrés fran. d'oto-rhino-laryng., Paris **10:** 15, 1930.
52. Gilfoy: Arch. Otolaryng. **16:** 182, 1932.
53. Guisez: Bull. d'oto-rhino-laryngol. **18:** 9, 1919 (3 cases). Congrès francais d'oto-rhino-laryngol., 1934.
54. Hajek: Wien. Laryng. Gesellsch. **7:** 2, 1929.
55. Hald: Soc. Dan. d'oto-laryng., April 3, 1912 (2 cases).
56. Hamacher: Inaug. Diss., Giessen, 1915.
57. Hama Tako: Otologia **3:** 515.
58. Harris and Forbes: Tr. Am. Laryng. A. **46:** 173, 1924.
59. Harssen: Med. Rev., October, 1887.
60. Heinzemann: Inaug. Diss., München, 1904.
61. Henrici: Arch. f. Laryngol. u. Rhinol. **17:** 283, 1905.
62. Heymann: Ztschr. f. Laryng., Rhin., Otol. **6:** 735, 1913.
63. Hinojar: Arch. de med., cir. y especialid. **36:** 1933.
64. Hinteroisser: Wien. klin. Wchnschr. **2:** 19, 374, 1889.
65. Hoffmann: Verhandl. d. Ver. Süddeutsch. Laryngol. **1:** 125, 1905.
66. Holmgren: Arch. f. Ohren-, Nasen-u. Kehlkopfh. **122:** 145, 1929 (3 cases).
67. Hug: Cor.-Bl. f. schweiz. Aerzte **1:** 47, 1919.
68. Iglauer: J. Med. **9:** 483, 1928.
69. Jacques and Hoche: Bronchoscop. Oesophagoscop. et Gastroscop. **3:** 234, 1934.
70. Joshida: Killian-Festschr. der Jap. oto-rhino-laryng. Gesellschr. 106, 1920.
71. Kahler: Soc. All. de Laryngol. **43:** 580, 1909 (2 cases).
72. Kaunitz: Wien. klin. Wchnschr. **38:** 1912 (3 cases).
73. Klebs: Virchows Arch. f. path. Anat. **38:** 212, 1867.
74. Koch: Ztschr. f. Wundärzte u. Geburtsch. **21:** 3, 184, 1868.
75. Körner: München. med. Wchnschr. **11:** 1888.
76. Koschier: Wien. klin. Wchnschr. **10:** 1006, 1897.
77. Krieg: Beitr. z. klin. Chir. **58:** 162, 1908.
78. Krompecher: Arch. f. Laryngol. u. Rhinol. **31:** 443, 1918 (2 cases).
79. Langhans: Arch. f. path. Anat., Berl. **53:** 470, 1871.
80. Lemaitre: Congrès francais d'oto-rhino-laryngol., 1934 (2 cases).
81. Leocini and Orlandi: Bull. d. clin. **1:** 10, 1922.
82. Leroux-Robert: Ann. d'oto-laryng. **5:** 493, 1936.
83. Link: Arch. f. Ohrenheilk. **126:** 262, 1930.
84. Litwinowicz: Soc. Med. de Temberg, M. F. O., 913, 1910.
85. Mackenzie: Krankh. d. Halses. Deutsch. v. Semon **1:** 711.
86. Maier: Deutsche Ztschr. f. Chir. **201:** 270, 1927.
87. Minningerode: Ztschr. f. Hals-, Nasen-u. Ohrenh. **9:** 37, 1924 (2 cases).
88. Morra: Gior. internaz. d. sc. md., Napoli **1:** 1129, 1879.
89. Nager: Arch. f. Laryngol. u. Rhinol. **20:** 275. **1907.**

90. Negus: Proc. Roy. Soc. Med. **26**: 238, 1933.
91. Nielson: J. Laryng. & Otol. **45**: 855, 1930.
92. Noehren and Kummer: Arch. Path. **2**: 508, 1926.
93. Oestereich: Deutsche med. Wchnschr. **21**: 34, 1895; Ztschr. f. klin. Med. **28**: 5 (3 cases).
94. Onodi: Arch. f. Ohren-, Nasen-u. Kehlkopfh. **3**: 236, 1917.
95. Oppikofer: Ztschr. f. Laryng., Rhin., Otol. **7**: 248, 1914.
96. Pick: Prag. med. Wchnschr. **16**: 69, 1891.
97. Pogrebinski: Yushno-Russk. Med. Gaz., Odessa **3**: 61, 1894.
98. Polayes: Proc. N. York Path. Soc. **25**: 100, 1925.
99. Proebsting: Vers. süddeutsch. Laryngol., 1896.
100. Puech: Montpellier med., July, 1888.
101. Rabuffetti and Others: Rev. Soc. de med. int. y. Soc. de tisiol. **7**: 473, 1931.
102. Reiche: Centralbl. f. allg. Path. u. path. Anat. **4**: 1, 1893.
103. Ricci: Arch. ital. di otol. **45**: 81, 1934 (2 cases).
104. Rokitansky: Ztschr. d. k. k. Ges. d. Aerzte z. Wien., 175, 1857.
105. Sabourin: Ann. d. mal. de l'oreille, du larynx **5**: 11, 1879.
106. Saltykow: Cor.-Bl. f. schweiz. Aerzte **41**: 673, 1911.
107. Schmidt: See von Bruns (Ref. 124).
108. Schmiegelow: Arch. f. Laryngol. u. Rhinol. **22**: 18, 1909.
109. Schrötter: Laryng. Mittheil 86, 1870; Jähresb. d. Klin. f. Laryngosk. **3**: 103, 1871; Anzeiger d. Ges. d. Aerzte in Wien. 9, 1877; Vorlesungen über d. Krankh. d. Luftröhre, 128 (4 cases).
110. Shattuck: Boston M. & S. J. **98**: 578, 1878.
111. Simmel: Arch. f. Laryngol. u. Rhinol. **24**: 449, 1910.
112. Simpson and Moore: Ann. Otol. Rhin. & Laryng. **43**: 1133, 1934.
113. Sörensen: Arch. f. Laryngol. u. Rhinol. **29**: 188, 1915.
114. Stenn: Arch. Otolaryng. **21**: 190, 1935.
115. Stenström: Acta med. Scandinav. **50**: 45, 1931.
116. Syme: J. Laryng. & Otol. **39**: 6, 305, 1924.
117. Teubert: Ztschr. f. Hals-, Nasen-u. Ohrenh. **33**: 444, 1933.
118. Theisen: Tr. 22nd. Meet. Am. Laryng. A. **38**: 264, 1906.
119. Tiling: Monatschr. f. Ohrenh. **67**: 322, 1933.
120. Torres de Azevedo: Mem. Inst. Oswaldo Cruz. **20**: 5, 1927 (2 cases).
121. Vinson and Leddy: Proc. Staff Meet., Mayo Clinic **8**: 641, 1933.
122. Virchow: Verhandl. d. Berl. med. Gesellsch. **18**: 188, 1888.
123. Vitrac: Arch. clin. de Bordeaux **5**: 573, 1896.
124. von Bruns: Handb. d. Laryngol. u. Rhinol. **1**: 2, 952, 1898.
125. von Meyenburg: Centralbl. f. allg. Path. u. path. Anat. **30**: 577, 1920.
126. von Schroetter: Klinik der Bronchoscopie, Jena 1906, 414 (2 cases).
127. Wadón: Centralbl. f. allg. Path. u. path. Anat. **60**: 308, 1934.
128. Wiethe: Monatschr. f. Ohrenh. **57**: 792, 1923.
129. Wildenberg and Dustin: Mém. Oto-rhino-laryng. internat. **8**: 5, 274, 1924.
130. Ziba: Arch. f. Laryngol. u. Rhinol. **25**: 401, 1911.

PRIMARY TUMORS OTHER THAN CARCINOMA SINCE 1929 (SEE TABLE I)

131. Abbate: Arch. f. Ohren-, Nasen-u. Kehlkopfh. **140**: 179, 1935.
132. Armand-Delille and Soulas: Bull. et mém. Soc. méd. d. hôp. de Paris **48**: 210, 1932.
133. Barth: Ztschr. f. Hals-, Nasen-u. Ohrenh. **30**: 583, 1932.
134. Beutel: Röntgenpraxis **6**: 287, 1934.
135. Biermann and Weiss: Ztschr. f. Laryng. Rhin. Otol. **22**: 311, 1932.
136. Bignami: Pathologica **27**: 665, 1935.
137. Elantsev: Sovet. vrach. zhur. 772, May 30, 1936.
138. Gatewood: Arch. Otolaryng. **24**: 92, 1936.
139. Hennell: J. Mt. Sinai Hosp. **2**: 30, 1935.
140. Jackson: J. A. M. A. **99**: 1747, 1932.
141. Ricci: Arch. ital. di otol. **45**: 81, 1934.
142. Richards and Dietrich: Ann. Otol. Rhin. & Laryng. **43**: 892, 1934.
143. Schöndorf: Internat. Zentralbl. f. Ohrenh. **37**: 1, 1933.
144. Soerensen and Schoetz: Berlin O.R.L. Gesellsch., 1933, 15.
145. Syme: J. Laryng. & Otol. **39**: 6, 305, 1924.
146. Zamora: J. Laryng. & Otol. **46**: 829, 1931.
147. Five unreported benign growths from the Department of Pathology, Johns Hopkins Hospital, Baltimore.
148. One unreported benign polyp from the Pathological Institute, McGill University, Montreal.

BRONCHIAL OBSTRUCTION PRODUCED BY ORGANIC AND INORGANIC FOREIGN BODIES

JOSEPH WEINBERG, M.D.
OMAHA, NEB.

THERE are two types of reactions following the intrabronchial lodgment of foreign bodies; a common one purely due to the matter of more or less complete bronchial occlusion, and a variable one depending on adventitious effects of the occluding body. Inert substances, particularly metals, often produce relatively mild early effects, and it may be months or years before they produce sufficient symptoms to make their presence known. In contrast to these, certain vegetable substances, of which the peanut is a notable example, may produce fulminating symptoms within a few hours of their lodgment. While these differences are generally appreciated, principally through the contributions and teachings of Chevalier Jackson, there are important points in the pathology which remain obscure.

In a previous study,[1] I described a method for the lodgment and retention of foreign bodies within the bronchus of the rabbit. This method is used in the present work to determine the early and late effects of representative types of foreign bodies, including the peanut, castor bean, nail, and tooth.

EXPERIMENTS

The foreign body is fitted into the flanged end of a ureteral catheter which is then introduced through a transtracheal opening into a bronchus. When resistance to further passage is encountered, the foreign body is dislodged from the catheter by means of a wire stylet. The catheter is then removed and the cervical incision is closed. This is done under ether anesthesia.

This study is based upon an analysis of eight to twelve successful experiments with each type of foreign body. The early and late results obtained are described in the following typical experiments:

EARLY CHANGES

Experiment W 1.—A fragment of castor bean was inserted in the bronchus of a rabbit. The rabbit died thirty-six hours after lodgment of the foreign body. At autopsy there was a thin, rather clear exudate in the bronchial tree. The foreign body was not found, and it is assumed that it was dislodged before death. There was a massive pneumonitis of the lower lobe of the left lung. The other lobes

Read before the Twentieth Annual Meeting of the American Association for Thoracic Surgery at Saranac Lake, N. Y., May 31, June 1 and 2, 1937.

showed a less severe pneumonitis, but in all lobes there was gross evidence of a violent inflammatory reaction associated with hemorrhages.

The left lower lobe revealed patchy hemorrhagic areas throughout the lung. There was marked edema of the parenchymal tissue and bronchi, with slight leucocytic infiltration. The mucosa of the bronchi showed marked degenerative changes and edema.

Sections of other lobes examined histologically revealed a marked edema and moderate congestion of the alveoli and bronchi together with moderate hemorrhage.

Because of the severe changes seen in the left lower lobe, it is assumed that the foreign body had lodged in the left lower bronchus.

In this experiment the castor bean provoked a fulminating reaction which was characterized by edema and hemorrhage. It is assumed that the ricinic acid of the castor bean is responsible for the severe reaction.

Experiment X 7.—A fragment of peanut was inserted into the bronchus of a rabbit. The rabbit died sixty hours later. Autopsy revealed a violent hemorrhagic inflammatory reaction in both lungs. Blood-tinged frothy fluid escaped from the tracheobronchial tree. All lobes showed hemorrhagic consolidation with occasional patches of aeration. There was more secretion from the left main bronchus than from the others. This was a thin, frothy, hemorrhagic fluid. A peanut was found lodged loosely in the left main bronchus near the carina.

The left lower lobe which was obstructed showed marked edema. The alveolar walls were barely distinguishable because of edema. There were numerous rounded dilated spaces in the parenchyma apparently due to rupture of the alveolar walls by distention through the valve action of the obstructing body. The parenchymal tissue was congested and hemorrhagic throughout. The bronchi were uniformly dilated and some of them contained purulent exudate (Fig. 1).

The right lower nonobstructed lobe showed marked edema, congestion and hemorrhage in the alveolar tissue with purulent exudate in the bronchial tree. The bronchi were only moderately dilated, and their mucosal linings were intact. In this specimen the alveolar walls were more distinct than they were in the specimen from the obstructed lobe.

It is assumed that in this experiment the pathologic changes in the obstructed lobe were the result of irritation by the peanut and, to a lesser degree, occlusion of the bronchus. This assumption is based upon the greater destruction and dilatation of the bronchi seen in the obstructed lobe. There was no evidence of swelling of the peanut.

Experiment X 1.—A peanut was lodged in the bronchus by the usual method. The rabbit was killed six days later. The peanut was found lodged loosely in the right main bronchus just below the carina. It was covered with mucous secretion. The trachea and bronchi were congested at this site.

There were gross petechial hemorrhages in the upper left lobe. The right lower lobe showed areas of patchy atelectasis.

Microscopic study of sections from the right lower lobe showed the bronchi to be dilated, with occasional interruptions of the mucosa. The blood vessels were greatly thickened, with evidence of marked edema in all coats. The alveoli were disrupted, congested, and infiltrated with leucocytes. There was moderate evidence of edema and patchy atelectasis.

Microscopic sections from the nonobstructed left lower lobe showed marked congestion, large areas of hemorrhage, edema, and thickening of the blood vessels. The bronchi were moderately dilated but most of them showed a fairly intact mucosa.

In this experiment there was marked edema in both the obstructed and nonobstructed lobes, suggesting that the reaction was the result

Fig. 1.

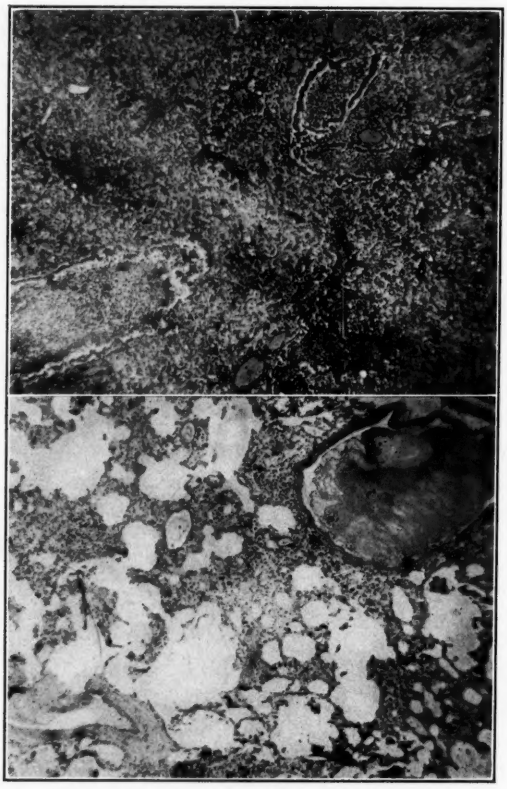


Fig. 2.

Fig. 1.—X7. Section of lung sixty hours after lodgment of fragment of peanut in a bronchus. The alveolar markings are obliterated by hemorrhage, congestion and edema, and the bronchi are filled with exudate. The changes were essentially the same in both lungs, although only one lobe was obstructed by the peanut. (×42.)

Fig. 2.—Z18. Section of obstructed lobe ninety hours after lodgment of a nail in the bronchus. The congestion and edema seen here are limited to the immediate site of lodgment of the nail. A comparison with Fig. 1 illustrates the mild character of the reaction in comparison to that of the peanut in the early stage of obstruction. (×42.)

of the spread of irritating oil to both lungs. The fact that the atelectasis was not marked in the lobe distal to the foreign body indicated that the obstruction was not complete, and that the pathologic changes were presumably mainly the result of the irritating action of the peanut oil.

Experiment Z 18.—A nail was lodged in the bronchial tree. The animal was killed four days later. Autopsy revealed a nail lodged loosely in the right main bronchus. There was an accumulation of mucous secretion in the right bronchus but none in the left.

Microscopic examination showed partial to complete loss of the mucosa of the bronchi in the lobe distal to the nail. The parenchymal tissue showed moderate edema and patchy atelectasis (Fig. 2). Also, there were petechial hemorrhages in the parenchyma, but less severe than those seen in the experiments with the peanut. The opposite lung showed very moderate changes characterized by congestion.

In this experiment the disturbances seem to have resulted chiefly from the mechanical presence of the nail. The hemorrhages may reasonably be ascribed to the disruption of vessels during lodgment. Thus, there were neither changes due to obstruction nor changes due to chemical irritation, the pathology being entirely the result of local mechanical disturbance.

Experiment Y 6.—An unsterilized rabbit tooth was lodged in the bronchial tree. The rabbit was killed forty-eight hours later. The tooth was found lodged in the right main bronchus. There were gross areas of hemorrhage in the left lower lobe. The right lower lobe showed extensive areas of patchy atelectasis and edema of the alveoli. The blood vessels were markedly thickened. Congestion and leucocytic infiltration were pronounced. Most of the bronchi and bronchioles showed loss of mucosa but were not greatly dilated. There were also frequent interruptions in the alveolar walls.

The left nonobstructed lobe showed dilatation of the bronchi with accumulation of purulent exudate. The mucosal linings were fairly intact. There were also small patches of atelectasis. Edema and congestion were present in the parenchyma to a moderate degree.

In this experiment, striking changes occurred within a forty-eight-hour period. The marked congestion and leucocytic infiltration of the alveolar tissue, and the loss of bronchial mucosa seen in the obstructed lobe indicate an early severe infectious process. It is assumed that the changes in the other lobes were due to the spread of infected secretion from the obstructed lobe and not to any toxic substance in the tooth, as was the case in the peanut and castor bean.

LATE CHANGES

Experiment X 5.—A fragment of peanut was lodged in the bronchial tree. The rabbit was killed twenty days later. Postmortem examination revealed a profuse exudate in the trachea. The foreign body, coated with exudate, was found at the first bifurcation of the right lower bronchus. The right lower lobe was adherent and contained multiple abscesses. The bronchi were grossly dilated and exuded pus. The lobe was congested and firm.

The nonobstructed lobes were congested but were otherwise normal grossly.

Microscopic study of the right lower lobe showed an advanced degree of bronchiectasis with almost complete atelectasis, abscesses throughout the parenchyma, dilated smooth contoured bronchi with loss of mucosa, and destructive changes in the bronchial walls. All the bronchi were filled with thick pus (Fig. 3).

Microscopic study of left lower lobe showed rather large patches of atelectasis, congestion and moderate dilatation of the bronchi. There was also evidence of edema in the nonatelectatic tissue.

The complete obstruction of the right lower lobe bronchus resulted in massive atelectasis and severe bronchiectasis in the right lower lobe. The patchy atelectasis and the inflammatory changes seen in the opposite lobe were the result of spread of the exudate from the obstructed lobe.

Fig. 3.

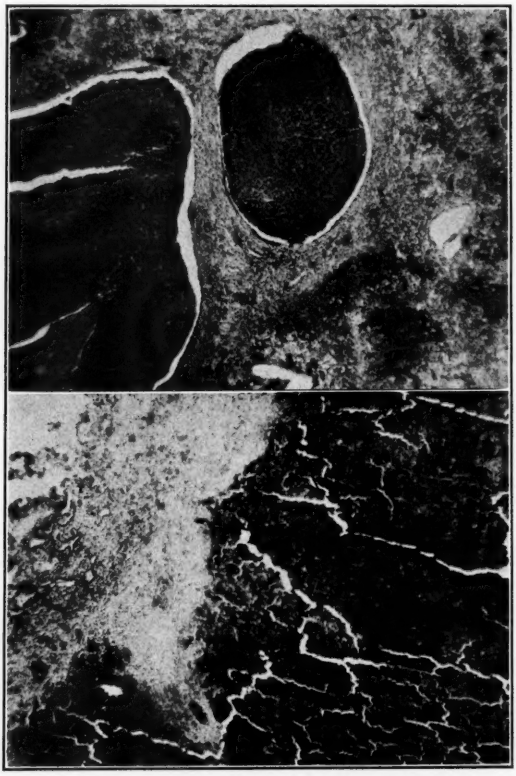


Fig. 4.

Fig. 3.—X5. Section of obstructed lobe twenty days after lodgment of fragment of peanut in a bronchus showing advanced saccular bronchiectasis. The dilated bronchi are filled with pus, and the alveolar tissue shows abscesses and complete atelectasis. (×42.)

Fig. 4.—Z6. Section of obstructed lobe forty days after lodgment of nail in a bronchus. This specimen represents an advanced stage of obstructive bronchiectasis with suppuration, atelectasis and fibrosis. The bronchial wall is not distinguishable, because of the severe destructive changes. (×42.)

Experiment Z 6.—A nail was lodged in the bronchial tree. X-ray examination made one week later showed the nail in the thorax, apparently in the bronchial tree. The animal was killed forty days later. There was a small amount of exudate in the trachea. The nail was not found at autopsy. The right lower and middle lobes showed multiple large abscesses through the pleura. These lobes were firm and congested. The main right lower bronchus was dilated and had a thickened

wall. Thick pus exuded from all cut surfaces of the middle and lower lobes. These lobes were adherent to the parietal wall.

Microscopic examination of the right lower lobe showed complete atelectasis with multiple abscesses in the parenchymal tissue. The bronchi could not be distinguished because of the advanced state of suppuration and necrosis (Fig. 4).

The left lower lobe showed comparatively slight evidence of inflammation. There was moderate congestion and leucocytic infiltration with serofibrinous secretion in the bronchial tree. The bronchi were not dilated.

In this experiment the left lower lobe shows the characteristic features of complete obstruction, including extensive atelectasis, destruction of bronchi, and parenchymal suppuration. Although it was not possible to retain the nail within the bronchus for a long period of time in this and other experiments, the changes found indicate that it had obstructed the bronchus at some time previous to the expulsion.

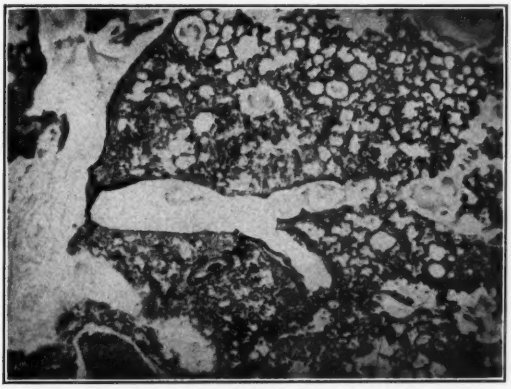


Fig. 5.—Y3. Section of incompletely obstructed lobe forty-eight days after lodgment of tooth. This section, taken from the immediate site of lodgment, shows a moderate cylindric bronchiectasis with little alteration of the parenchyma. (×42.)

Experiment Y3.—A rabbit tooth was lodged in the bronchial tree. The rabbit was killed forty-eight days later. The tooth was found lodged in the left main bronchus. There was an extreme congestion of the bronchus at the site of its lodgment. The left lower lobe showed gross evidence of patchy atelectasis and all other lobes were congested. A frothy exudate was found in the tracheobronchial tree. Pus exuded from the cut surface of the left lower lobe. There were occasional abscesses in the parenchyma of the right lower lobe.

The tooth which was in a good state of preservation was only slightly coated with exudate. The obstruction was incomplete.

Microscopic examination of the partially obstructed lobe showed remarkably little change considering the long period of time that the tooth had remained in the bronchial tree. The bronchi were moderately dilated and showed loss of mucosa and destructive changes in their walls (Fig. 5). There were occasional accumulations of purulent material in the alveolar spaces and bronchial tree. One area of the lobe showed a solitary abscess.

Sections from other lobes showed much the same changes as were seen in the partially obstructed lobe but to a lesser degree. Congestion and moderate edema were the principal changes although there was evidence of moderate dilatation of the bronchi.

It would seem from this experiment that the long sojourn of the tooth in the bronchus failed to induce severe pathologic changes. In all probability this is to be accounted for by the obstruction being incomplete rather than complete. Here the foreign body has apparently acted chiefly as an irritant with a possible added factor of infection through organisms carried with it.

The findings in the experiments with the sandbur, which have been reported previously,[1] are included in the discussion which follows.

DISCUSSION

The experimental results are discussed under the headings of (1) immediate effects produced by the various types of foreign bodies and (2) the late effects, with a consideration of the findings in complete and incomplete obstruction.

1. The type of pathologic change produced in the lung within the first few days after lodgment depends principally upon the chemical nature of the foreign body. Vegetable substances containing soluble irritants, represented in these experiments by the castor bean and peanut, produce within the first forty-eight hours a fulminating reaction in both lungs characterized by edema and hemorrhage, the former especially causing interference with respiration. This reaction was most violent in the case of the castor bean, death occurring within a two-day period in all instances in which the substance was used. The reaction with the peanut was of the same nature but it was less violent. In those instances in which the animal succumbed to the immediate reaction, death occurred within a three-day period. In addition to the edema and hemorrhage there was evidence of degeneration of bronchial mucosa and dilatation of the bronchi in the obstructed lobes (Fig. 1). In no instance was there evidence of swelling of the peanut or castor bean after lodgment in the bronchus. In fact, the fragment was lodged rather loosely at the time of autopsy. This point is emphasized because frequently it is stated without adequate evidence that the severe symptoms from peanut aspiration are chiefly due to the swelling after its lodgment. The chief factor would appear to be the soluble toxic fraction in both these foreign bodies, ricinic acid in the case of the castor bean and arachidic acid in the case of the peanut. This is in accord with the experimental findings of Heatley and Clausen,[2] Pinkerton[3] and Hara,[4] and with the clinical observations of Jackson,[5] Jackson and Spencer,[6] Patterson,[7] and others. This highly irritating effect is not peculiar to all vegetable substances. In experiments which

I have previously performed with the sandbur there was no evidence of edema, and the slight hemorrhages that occurred were obviously due to disruption of the bronchial wall at the site of lodgment. In view of this, it is doubtful whether a distinction between vegetable and nonvegetable foreign bodies based on differences in irritating properties is justifiable. The difference is rather one of whether or not the foreign body contains an irritating soluble fraction which may be diffused through the lungs.

Inert substances such as the tooth and nail provoke a less severe type of reaction within the first few days than do the peanut and castor bean. In the case of the nail (Fig. 2) the pathologic change, consisting chiefly of local hemorrhage and ulceration of the bronchial mucosa, may be accounted for by the trauma occurring during and after lodgment.

The reaction to the tooth in the early stage is, in some cases, mild and entirely local and is the result of mechanical irritation at the site of lodgment. In other instances it is characterized by striking changes including patchy atelectasis, purulent exudate in the obstructed and nonobstructed lobes, and petechial hemorrhages. These changes are predominantly infectious and do not seem to depend to a great extent upon obstruction. There is nothing to indicate that the cause of the disturbance is of a chemical nature. It is possible that the type and degree of reaction are influenced by the number and virulence of organisms carried with the tooth.

2. The late effects of bronchial occlusion, whether by irritating vegetable substances or by inert substances, appear to depend chiefly upon the completeness of obstruction, the presence or absence of a soluble toxic fraction playing little if any rôle at this stage. If the obstruction is complete, there will result a massive atelectasis distal to the occlusion, together with suppurative infection and saccular dilatation of the bronchi (Figs. 3 and 4). The mechanism of the alterations with complete obstruction is not described in detail here, as it has been dealt with in a previous study.[1] Experiments with varying periods of sojourn indicate that the obstruction is usually incomplete at first, but may eventually become complete through the accumulation of fibrinous exudate around the foreign body. Following this, the entire lobe becomes atelectatic,* and this together with the degeneration and necrosis resulting from retained infectious secretions causes a severe saccular bronchiectasis. The nonobstructed lobes also undergo changes which are characterized by cylindric dilatation of the bronchi, and patchy areas of atelectasis and suppuration, due to the passage of infectious exudate from the obstructed lobe into the bronchi of other lobes. It should be emphasized that it is not neces-

*The reader is referred to the researches of Lichtheim,[8] and Coryllos and Birnbaum,[9] for a detailed explanation of atelectasis resulting from bronchial occlusion.

sary to assume the introduction of pathogenic microorganisms with the foreign body to produce infection. Evidence indicates that the lung normally harbors potentially pathogenic bacteria which become activated in the retained secretion distal to the foreign body. (Bucher,[10] Jones,[11] Boni,[12] Neisser,[13] Spray,[14] Livingstone and Adams.[15])

If the obstruction remains incomplete, the late changes vary from a local ulceration of the bronchus at the site of lodgment to an advanced cylindric bronchiectasis which may approach the obstructive type of bronchiectasis in severity. In these experiments the tooth was the only foreign body which did not tend to cause a complete obstruction after a lapse of several weeks. Possible explanations for this peculiarity are the irregular shape of the tooth and its lack of tendency to become coated with fibrinopurulent exudate. Lodgment of the tooth, however, is capable of producing severe pathologic changes through other mechanisms. The bronchus becomes ulcerated at the site of lodgment, as a result of trauma, and the spread of infectious secretion from this site into the general bronchial tree causes cylindric bronchial dilatation, patchy areas of atelectasis, and infection varying from a mild inflammation to extensive abscess formation.

SUMMARY AND CONCLUSIONS

A series of experiments have been performed in which several types of foreign bodies have been lodged in the bronchial tree for varying periods of time, and the pathologic effects noted. In these experiments the manner of lodgment and the types of foreign bodies permit a comparison with the conditions obtaining in clinical practice.

Certain vegetable foreign bodies, of which the peanut is representative, produce immediate fulminating edema and hemorrhage in the lungs which may cause death within the first few days of lodgment. It would appear that the reaction is due to irritating substances released by these foreign bodies, and that the obstruction plays little part in the early disturbance.

In the case of more inert substances, represented by the nail and tooth in these experiments, the changes seen in the first few days following lodgment are less severe, and usually consist of degeneration and ulceration at the site of lodgment, due to trauma induced during and after lodgment. Occasionally there is an additional change characterized by suppuration and patchy atelectasis in the obstructed and other lobes. It is probable that this is due to, or accelerated by, pathogenic organisms introduced with the foreign body.

The late effects of bronchial occlusion show a striking similarity to each other, and appear to depend chiefly upon the degree of obstruction rather than upon the composition of the foreign body. This is in contrast to the varied reactions produced by irritating vegetable sub-

stances and inert substances in the early stage of lodgment. Complete obstruction eventually causes massive atelectasis and severe saccular bronchiectasis.

If the obstruction remains incomplete for a relatively long period, the pathologic changes may be slight or severe depending upon the degree of obstruction, upon the trauma induced by the foreign body, and upon the pathogenicity of infectious organisms introduced or already present. The characteristic pathologic changes seen with incomplete obstruction are patchy atelectasis, suppuration, and bronchial dilatation.

REFERENCES

1. Weinberg, J.: Experimental Production of Bronchiectasis, J. THORACIC SURG. **6**: 402, 1937.
2. Heatley, C. A., and Clausen, S. W.: Experimental Studies in Peanut Bronchitis, Arch. Otolaryng. **11**: 569, 1930.
3. Pinkerton, H.: Reaction to Oils and Fats in the Lung, Arch. Path. **5**: 380, 1928.
4. Hara, H. J.: Organic Foreign Bodies in the Bronchi, Arch. Otolaryng. **20**: 549, 1934.
5. Jackson, C.: Peroral Endoscopy and Laryngeal Surgery, St. Louis, Laryngoscope Co.
6. Jackson, C., and Spencer, W. H.: Arachidic Bronchitis, J. A. M. A. **73**: 672, 1919.
7. Patterson, E. J.: Peanut Bronchitis, New York State J. Med. **109**: 101, 1919.
8. Lichtheim, L.: Experiments Dealing With Pulmonary Atelectasis, Arch. f. exper. Path. u. Pharmakol. **10**: 54, 1879.
9. Coryllos, P. N., and Birnbaum, G. L.: Obstructive Massive Atelectasis of Lung, Arch. Surg. **16**: 501, 1928.
10. Bucher, C. J.: Bacterial Flora Associated With Foreign Bodies in Trachea and Bronchi, Am. J. M. Sc. **190**: 409, 1935.
11. Jones, F. S.: Source of Micro-organisms in Lungs of Normal Animals, J. Exper. Med. **36**: 317, 1922.
12. Boni, I.: Untersuchung uber den Keimgehalt der normalen Lungen, Deutsches Arch. f. klin. Med. **69**: 542, 1901.
13. Neisser, M.: Ueber die Duchgangigkeit der Darmwand fur Bakterien, Ztschr. f. Hyg. u. Infectionskr. **22**: 12, 1896.
14. Spray, R. S.: Bacteria in Normal and Diseased Lungs of Swine, J. Infect. Dis. **31**: 10, 1922.
15. Livingstone, H., and Adams, W. E.: Bacterial Flora of Respiratory System, J. Infect. Dis. **48**: 282, 1931.

UNEXPECTED CAVITY CLOSURE FOLLOWING REEXPANSION OF THE LUNG AFTER AN INEFFECTUAL PNEUMOTHORAX*†

JOHN D. STEELE, JR., M.D., MILWAUKEE, WIS., JOHN W. TRENIS, M.D., ANN ARBOR, MICH., AND EDWARD W. LABOE, M.D., HOWELL, MICH.

CLOSURE of a tuberculous cavity following reexpansion of the lung after an ineffectual pneumothorax is a paradoxical occurrence which we have had the occasion to observe several times during the past year. Since we believed that cavity closure under these circumstances was important enough to warrant further investigation, we have collected a series of 12 cases in which such cavity closure has occurred and have analyzed them in detail.

In most instances it had been the intention to replace the ineffectual pneumothorax with some other form of collapse therapy, such as thoracoplasty, and the lung had been allowed to reexpand in preparation for that procedure.

We attempted to estimate the frequency of such cavity closure by comparing our patients with a group in which the cavities failed to close following reexpansion of the lung. We found, however, a variety of complicating factors which made comparison impossible. For instance, until two or three years ago, it had been our practice to perform a thoracoplasty without waiting for the absorption of an ineffectual pneumothorax. This necessarily eliminated a large group of cases which would have formed a good basis for comparison.

Adhesions probably accounted for the failure of the pneumothorax to close the cavities in the majority of these patients. In retrospect, it appears to us that a longer trial of pneumothorax or an attempted division of the suspending adhesions might have been preferable to discontinuation of the pneumothorax in a few cases. We are, however, not concerned with the consideration of such possibilities in this article.

Cavity closure following reexpansion of the lung occurred more rapidly and more dramatically in those cases in which a phrenic paralysis was added at the time of discontinuation of the pneumothorax. Maximum elevation of the hemidiaphragm with resulting increase in pulmonary collapse is furthered by the higher negative pressures accompanying reexpansion of the lung. Phrenic paralysis is not as effectual if delayed until the lower lobe has become adherent and the hemidiaphragm relatively fixed by adhesions.

*From the Departments of Surgery and Internal Medicine, University of Michigan, Ann Arbor, Michigan, and from the Michigan State Sanatorium, Howell, Michigan.

†The preparation of this article was aided by the George Alexander Fleming Fund for Thoracic Surgery.

Received for publication, September 15, 1937.

In 7 of our 12 patients, phrenic nerve interruptions were performed at the time of discontinuation of the pneumothorax, and in one patient, three months before discontinuation. Cavity closure occurred without the addition of a phrenic paralysis in 2 patients (Cases 1 and

Fig. 1. Fig. 2.

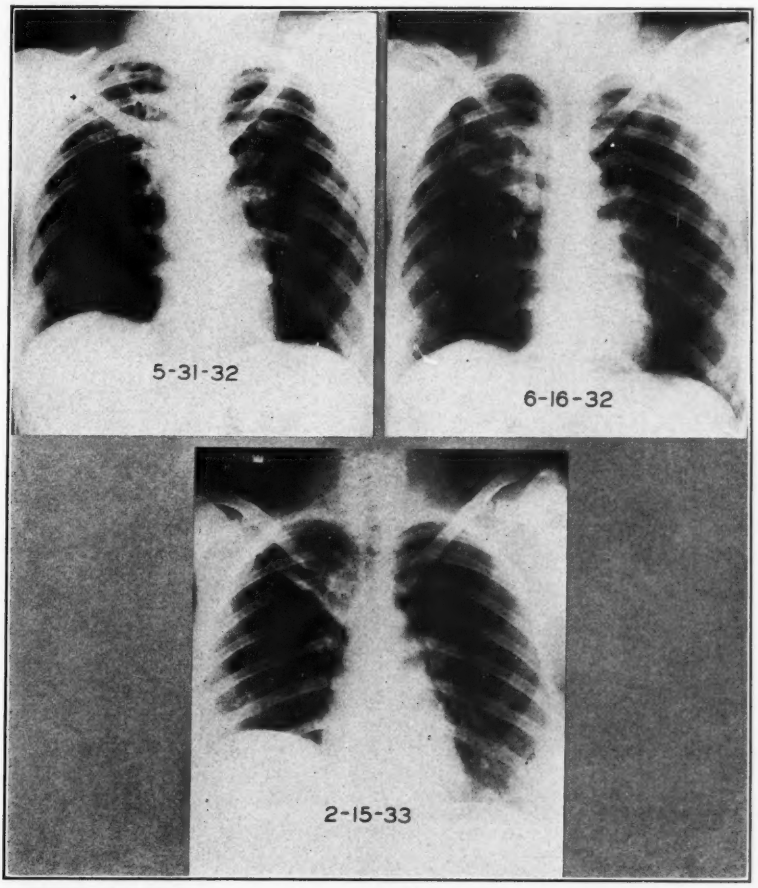


Fig. 3.

Fig. 1.—Case 3. Roentgenogram on admission. Cavity in right superior lobe 4.5 cm. in diameter.

Fig. 2.—Case 3. Roentgenogram following induction of right pneumothorax. Cavity reduced in size. Superior lobe suspended by adhesions.

Fig. 3.—Case 3. Roentgenogram following right temporary phrenic nerve interruption and discontinuation of pneumothorax. Marked regression of lesion. No evidence of cavity. Moderate rise of right hemidiaphragm.

5). In 2 other patients (Cases 8 and 10), a remarkable decrease in the size of the cavity accompanied reexpansion of the lung and cavity closure subsequently followed phrenic paralyses performed several

months after complete absorption of the pneumothorax. It is possible that more rapid closure of the cavities would have occurred in these 2 patients if the phrenic paralyses had been performed at the time of discontinuation of the pneumothorax and before the hemidiaphragm had become somewhat fixed by adhesions.

It seems probable that cavity closure in our group of patients was not necessarily the result of phrenic paralysis; first, because we have

Fig. 4. Fig. 5.

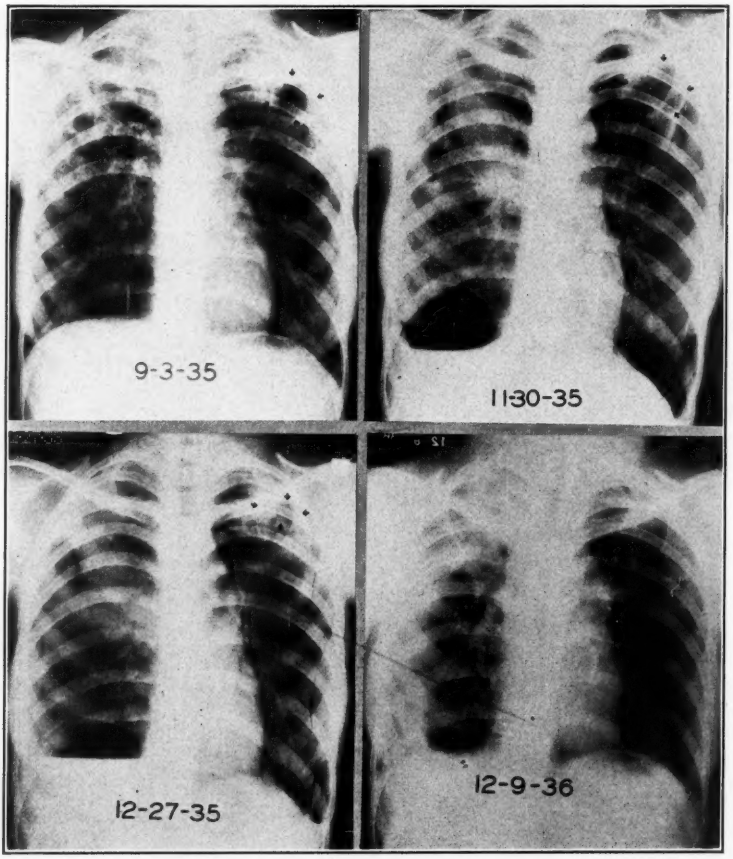


Fig. 6. Fig. 7.

Fig. 4.—Case 8. Roentgenogram on admission. Cavity in left superior lobe 3 cm. in diameter. Multiple cavities right superior lobe.

Fig. 5.—Case 8. Roentgenogram after induction of bilateral pneumothorax. Left superior lobe suspended by adhesions. Cavity has increased in size to 4.5 cm. in diameter. Cavities on right not evident.

Fig. 6.—Case 8. Roentgenogram sixteen days after discontinuation of left pneumothorax and seven days after discontinuation of right pneumothorax. Left lung almost completely reexpanded. Cavity now diminished markedly in size.

Fig. 7.—Case 8. Roentgenogram one year later. No evidence of cavity on either side. Sputum negative by culture.

seen cavities close rapidly following reexpansion of the lung when no phrenic paralysis had been added, and, second, because most of the cavities were too large to have been expected to close by phrenic paralysis alone in a corresponding period of time. The rapidity of cavity closure in these cases was the most impressive feature.

Fig. 8. Fig. 9.

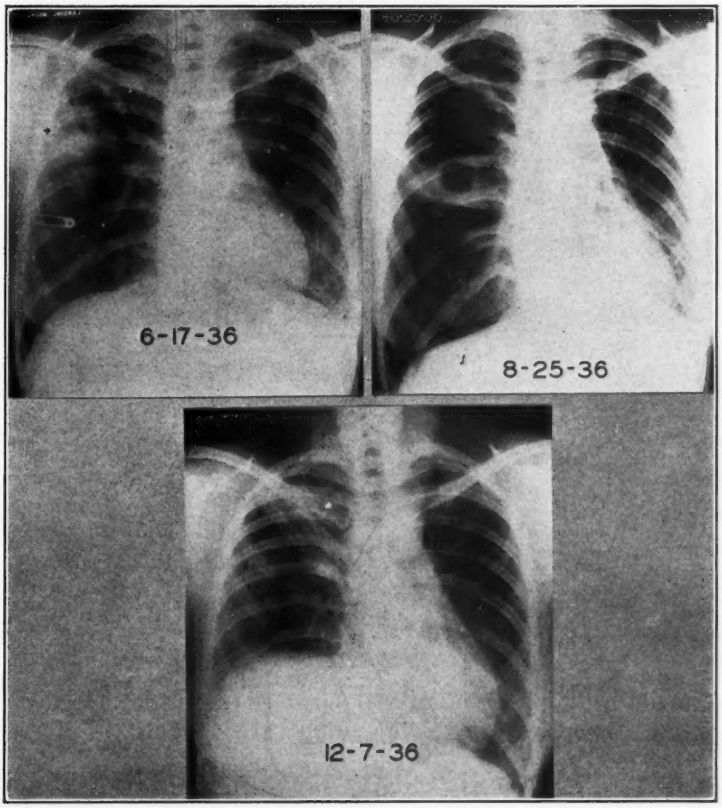


Fig. 10.

Fig. 8.—Case 12. Roentgenogram on admission. Cavity on right, 3.5 cm. in diameter.

Fig. 9.—Case 12. Roentgenogram two months after induction of right pneumothorax. Cavity now 2.5 cm. in diameter. Closure prevented by suspending adhesions. Right temporary phrenic nerve interruption two days later.

Fig. 10.—Case 12. Roentgenogram following discontinuation of pneumothorax. No evidence of cavity. Three and one-half months interval. Moderate elevation of right hemidiaphragm. Sputum negative.

Pleural effusion accompanying reexpansion was not prominent in most of our patients and probably had no influence upon cavity closure. Reexpansion in only one case was forced by obliterative pleuritis.

TABLE I

ANALYSIS OF CASES

CASE NUMBER AND INITIALS	AGE	SEX	DATE OF ONSET OF PULMONARY TUBERCULOSIS	DATE AND PLACE OF HOSPITALIZATION*	DATE OF INITIAL PNEUMOTHORAX	DATE OF LAST PNEUMOTHORAX REFILL	DATE OF TEMPORARY PHRENIC NERVE INTERRUPTION	ESTIMATED TIME INTERVAL BETWEEN LAST REFILL AND CAVITY CLOSURE	INTERVAL FROM LAST REFILL TO FIRST NEGATIVE SPUTUM†	DATE OF DISCHARGE FROM SANATORIUM	N.T.A. CLASSIFICATION AT TIME OF LAST OBSERVATION OR STATUS AS OF JUNE 1, 1937	REMARKS
Case 1 M. K.	18	F	May 1925	8/28/29 U. M. H.	Left 1/21/30 Right 3/13/30	Right 11/ 4/30	10/30/31	8½ mo.	3 mo. (1)	8/ 3/32	Unknown	Left open pneumonolysis on 11/8/30. Right permanent phrenic nerve interruption on 12/30/33. Thoracoplasty or paraffin pneumonolysis had been intended after reexpansion of right lung.
Case 2 L. P.	26	F	1925	12/22/30 M. S. S.	Right 12/30/30	2/18/31	2/18/31	10 mo.	11 mo. (2)	10/15/32	Active 1936	Developed new lesion in right posterior lobe following pyelolithotomy in August, 1934. Right extrapleural paraffin pneumonolysis in September, 1934. Sputum still positive although original cavity is closed.
Case 3 H. W.	16	F	Feb. 1932	6/ 8/32 J. C. S.	Right 6/13/32	6/16/32	6/23/32	8 mo.	3 mo. (1)	11/ 2/33	Apparently cured 1937	Pneumothorax discontinued after two refills even though cavity had become smaller.
Case 4 F. H.	43	F	Dec. 1932	4/29/33 U. M. H.	Left 5/ 8/33	10/ 4/33	6/13/33	3 mo.	4 mo. (2)	1/23/35	Arrested 1935	Pneumothorax discontinued because of obliterative pleuritis.

*Key to Sanatoria:
M.S.S.—Michigan State Sanatorium, Howell, Michigan.
J.C.S.—Jackson County Sanatorium, Jackson, Michigan.
I.C.S.—Ingham County Sanatorium, Lansing, Michigan.
U.M.H.—University of Michigan Hospital, Ann Arbor, Michigan.

†Key to sputum examinations:
(1)—Direct smear.
(2)—Concentrate.
(3)—Culture.

TABLE I—CONT'D

Case 5 R. H.	31	M	1927	1/20/34 M. S. S.	Right 3/ 7/34	11/11/34	None	5½ mo.	5 mo. (3)	6/19/36	Arrested 1937	On completion of a left thoracoplasty in November, 1934, the right pneumothorax was abandoned with the intention of performing a paraffin pneumonolysis after reexpansion of the lung.
Case 6 E. S.	38	F	Aug. 1933	5/ 5/34 M. S. S.	Right 5/ 7/34	11/12/34	11/ 6/34	4½ mo.	4 mo. (1)	11/13/36	Apparently arrested 1937	A marked elevation of the right hemidiaphragm followed the phrenic nerve interruption but cavity did not become smaller until after the pneumothorax was abandoned.
Case 7 C. T.	47	M	July 1933	7/ 6/34 J. C. S.	Right 7/16/34	7/28/34	8/ 3/34	4 mo.	5 mo. (1)	2/ 8/36	Arrested 1937	Cavity increased in size following induction of pneumothorax. Phrenic nerve interruption subsequently made permanent on 11/15/35.
Case 8 A. J.	20	F	Jan. 1935	8/30/35 U. M. H.	Left 9/16/35 Right 10/10/35	Left 12/11/35 Right 12/20/35	Left 7/21/36	10 mo.	10 mo. (3)	Resident M. S. S.	On increasing exercise	Cavity increased in size following induction of pneumothorax and diminished rapidly in size upon reexpansion of the lung.
Case 9 E. S.	34	F	Sept. 1932	9/18/35 J. C. S.	Right 9/20/35	1/16/36	3/20/36	4 mo.	8 mo. (1)	Resident J. C. S.	Quiescent	Right temporary phrenic nerve interruption repeated 12/30/36.
Case 10 E. C.	22	F	Dec. 1935	6/ 3/36 M. S. S.	Right 6/ 8/36	6/19/36	11/ 3/36	9 mo.	Remains positive	Resident M. S. S.	Active 1937	Cavity became much smaller following reexpansion of the lung and closed following the phrenic nerve interruption. Large cavity in contralateral lung unchanged in size since admission.
Case 11 G. S.	32	F	April 1936	6/10/36 U. M. H.	Left 6/22/36	9/28/36	10/13/36	4 mo.	None 2 mo.	Resident M. S. S.	On increasing exercise	Thoracoplasty intended when pneumothorax abandoned. Phrenic nerve interruption performed for hemoptysis during reexpansion.
Case 12 U. C.	29	F	Sept. 1931	6/17/36 I. C. S.	Left 6/20/36	8/25/36	8/27/36	3½ mo.	4 mo. (2)	Resident I. C. S.	Quiescent	Thoracoplasty intended when pneumothorax abandoned.

We had hoped that we could reach some conclusion as to the mechanism of cavity closure in these patients and that we might be able to find some common characteristic which would enable us to predict the outcome in similar cases. With these considerations in mind, we carefully studied a group of cases in which the cavities had failed to close following reexpansion of the lung. We were unable to reach any satisfactory conclusion as to the mechanism or to find any common char-

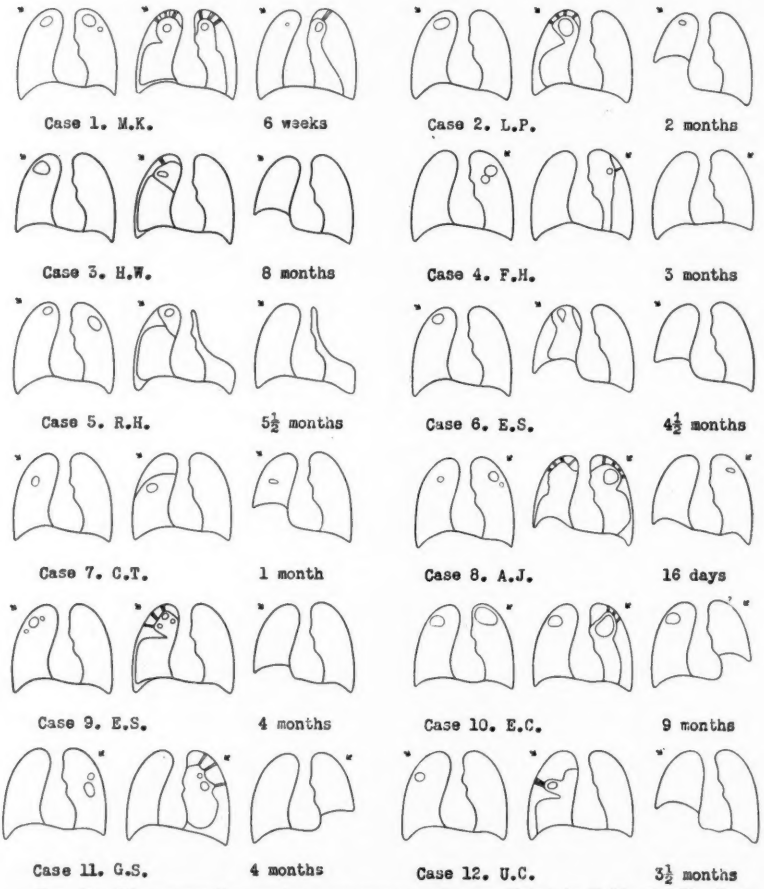


Fig. 11.—Diagrammatic roentgen appearance of cases listed in Table I. The first diagram in each series represents the appearance of the cavity, or cavities, immediately preceding induction of pneumothorax. The second diagram in each series represents the appearance at the time of maximum collapse by pneumothorax. The third diagram in each series represents the appearance after reexpansion of the lung. The period of time from the last pneumothorax refill is indicated below the diagrams.

These diagrams are intended to demonstrate the relative size of the cavities only. No attempt has been made to indicate noncavernous infiltration or to indicate accurately the character and extent of the visceroparietal adhesions. For this reason, some adhesions may appear to be easily divisible, while actually this was not the case. Sometimes the lung was fused to the anterior or posterior chest wall. This fusion could not be represented in the diagrams.

acteristic, such as the change of position of the cavity under pneumothorax, or any particular distribution or location of the collapse, which would aid us in predicting the outcome in similar cases in the future.

In conclusion we feel that cavity closure following absorption of an ineffectual pneumothorax probably occurs frequently enough so that this possibility should influence the decision as to whether to reexpand the lung before performing a thoracoplasty. Whenever not contraindicated, a temporary phrenic nerve interruption should be performed at the time the pneumothorax is discontinued and the benefit of its action determined before the thoracoplasty is performed. Marked decrease in the size of the cavity when reexpansion is complete warrants a period of delay, at the end of which there may be some indication as to whether final closure will occur without surgical intervention.

Three case reports are included in this article through the courtesy of Dr. R. J. Hanna, Superintendent of the Jackson County Sanatorium, Jackson, Michigan, and one case through the courtesy of Dr. G. C. Stucky, Superintendent of the Ingham County Sanatorium, Lansing, Michigan. The remaining cases were observed at the Michigan State Sanatorium, Howell, Michigan, and at the University of Michigan Hospital, Ann Arbor, Michigan.

OUR EXPERIENCES IN THE EMPLOYMENT OF SPINAL ANESTHESIA FOR THORACOPLASTY*

FRASER B. GURD, M.D., A. M. VINEBERG, M.D., AND
WESLEY BOURNE, M.D.
MONTREAL, CANADA

THAT surgeons who have made the surgical treatment of pulmonary tuberculosis a major interest are not satisfied that the most effective and safest anesthetic has yet been decided is proved by the fact that in different clinics different techniques for anesthesia are employed. Recent articles by Overholt and by Coryllos are significant in this respect.

That the majority of patients requiring thoracoplasty for pulmonary tuberculosis are not good surgical risks is so axiomatic that no further reference to this fact will be made here. As Shields[1] has stated, "In the presence of pulmonary tuberculosis spinal anesthesia would be chosen by many, should they be faced with an abdominal operation."

We believe that many, if not most, surgeons and anesthetists who have had any considerable experience in the employment of spinal anesthesia for upper abdominal operations—biliary tract, stomach, spleen, and hepatic and splenic flexures—and who are also interested in the surgery of pulmonary tuberculosis have wished that it were possible safely to employ spinal anesthesia in the performance of extrapleural thoracoplasty. Shields[2] has indicated that in the Toronto clinic spinal anesthesia has been employed in a large number of cases (55 up to 1935) for thoracoplasties from the 5th rib downward. He states (1933), "In this connection it should be remembered that a block which will produce adequate muscular relaxation for a gall bladder operation will give ample anesthesia for a lower thoracoplasty, with less circulatory depression as a rule in the extraabdominal operation."

Bettman and Biesenthal,[3] in 1931, reported five cases of extrapleural thoracoplasty performed under spinal anesthesia. They employed novocaine dissolved in cerebrospinal fluid, and injected the solution with the patient lying on the side to be operated upon. We do not know whether these authors have continued the use of spinal anesthesia. In 1935, Newton[4] employed a similar technique in twenty-eight operations. As preliminary medication he used morphine and scopolamine but in doses considerably smaller than we have employed. He injected ephedrine ten or fifteen minutes prior to induction of the spinal anesthesia.

*From the Grace Dart Home Hospital (for Pulmonary Tuberculosis).
Read (by title) before the Twentieth Annual Meeting of the American Association for Thoracic Surgery at Saranac Lake, N. Y., May 31, June 1 and 2, 1937.

Newton sums up his report by the statement, "I do not believe in using spinal anesthesia as a routine in thoracoplasty but only in patients with considerable mental poise."

We believe that there are sufficient reasons for desiring to use spinal anesthesia for thoracoplasty if a satisfactory and safe method can be employed. Our reason for reporting our experiences in this connection before this Association is in order to describe a technique which in our hands in but a limited number of cases, it is true, has proved eminently satisfactory.

During the past four years at the Grace Dart Home Hospital in Montreal, we have employed various combinations of anesthesia in the performance of thoracoplasties for pulmonary tuberculosis. In this way, we have used avertin, nembutal, local novocaine infiltration and nerve blocking, nitrous oxide and oxygen, cyclopropane, and intravenous evipal. It is not our intention, at the present time, to compare the results obtained by these different drugs, alone or in combination, but to express our opinion that since the employment of spinal anesthesia we have been impressed by both the comparative ease with which operation is performed and the more favorable results obtained.

Between July, 1936, and March, 1937, nine patients have been operated upon and seventeen operations performed under spinal anesthesia. As a rule, the cases have been divided in so far as operative procedures were concerned into the following: first stage, that is removal *in toto* of the 1st, 2nd and 3rd ribs together with their transverse processes; second stage, removal of the 4th, 5th and 6th ribs, together with their transverse processes, as far forward as the mid or anterior axillary line; the third stage has consisted in either simple removal of the 7th rib which has been accomplished by means of local novocaine infiltration or, more usually, the 7th, 8th and 9th ribs. Of the patients operated upon under spinal anesthesia, five were first stage, seven were second stage, and five third stage. Four of the nine patients had, prior to our use of spinal anesthesia, been operated upon for first or second stages using avertin as a basal anesthetic and cyclopropane.

There has been no mortality, nor has operation in any case been followed by a period, so far as we were concerned, of anxiety for the patient's welfare. The patients have uniformly been perfectly willing to undergo further operations, and have not been unduly apprehensive, less so, we believe, than has been the case with patients operated upon by other techniques. In no case has a transfusion been deemed necessary or advisable.

The technique described by Etherington-Wilson has been employed in all cases. As a preliminary sedative, morphine and scopolamine have been administered. Ephedrine has not been employed as a routine, and we consider this deviation from the more usual technique in the induction of spinal anesthesia to be of importance since the conserva-

tion of blood is thereby obtained. Etherington-Wilson[5] described his technique before the Royal Society of Medicine in 1933. Since that time further contributions[6] have been made by him, and the present status of the method was modernized by Bourne and O'Shaughnessy[7] in 1936. The essentials of the method are described herewith.

The patient is supported sitting upright, nupercaine in 1:1500 solution is employed for anesthesia, the needle introduced in the 2nd or 3rd lumbar interspace, and the solution injected slowly without barbotage. At the completion of the injection a stop watch is started and time allowed for the passage upward of the solution. Prior to our work in employing this method for top stage thoracoplasty it had been established that, depending upon the amount of solution injected and the time allowed for its passage upward, three main zones of analgesia could be obtained:

(1) 10 c.c. nupercaine solution (1:1500) 20 to 25 seconds—first lumbar nerve segments suitable for operations upon the lower limbs and the perineum.

(2) 12 c.c. nupercaine solution (1:1500) 30 to 35 seconds—tenth thoracic segments suitable for operations upon the lower abdomen and pelvis.

(3) 15 c.c. nupercaine solution (1:1500) 40 to 45 seconds—a high zone, fifth thoracic segments suitable for operations upon the upper abdomen and lower thorax.

As the result of our experiments we feel that we are able to say that:

(4) 15 to 17 c.c. nupercaine solution (1:1500) 55 to 60 seconds—lower cervical segments and suitable for operations up to and including the first rib.

At the end of the allotted number of seconds the upright position is quickly changed to that of recumbency, and the head of the table is tilted slightly downward. It would seem probable that an absolute maximum for the time that the patient may be allowed to sit upright is 65 seconds, and that the maximum quantity of the solution should be 17 c.c.

In our patients there have been no headaches or other unfavorable incidents or sequelae.

With respect to preliminary medication, we have found that the quantities of the drugs customarily used are not sufficient properly to obtund the cerebral cortex, and so to produce an optimum of bemusement and amnesia. With increased doses nausea does not occur and there is less shock. We believe that it is well thoroughly to subdue the activity of the highest nerve centers. We have come to prefer the use of morphine, gr. $\frac{1}{2}$, with scopolamine, gr. $\frac{1}{50}$, and atropine, gr. $\frac{1}{150}$, given hyperdermically all at once, one-half hour before operation.

Analeptics, such as ephedrine, have not been used routinely for two reasons: first, that they invariably undo to a considerable extent the

desired depressant effects of the hypnotic and amnesic drugs employed and, second, they are apt to raise the blood pressure so that troublesome oozing takes place. We do not, therefore, administer analeptics unless there is an alarming fall of blood pressure, at which time they seem to have their best actions. In accordance with the teachings of Yandell Henderson,[8] at the end of an operation $\frac{1}{30}$ gr. of strychnine is given

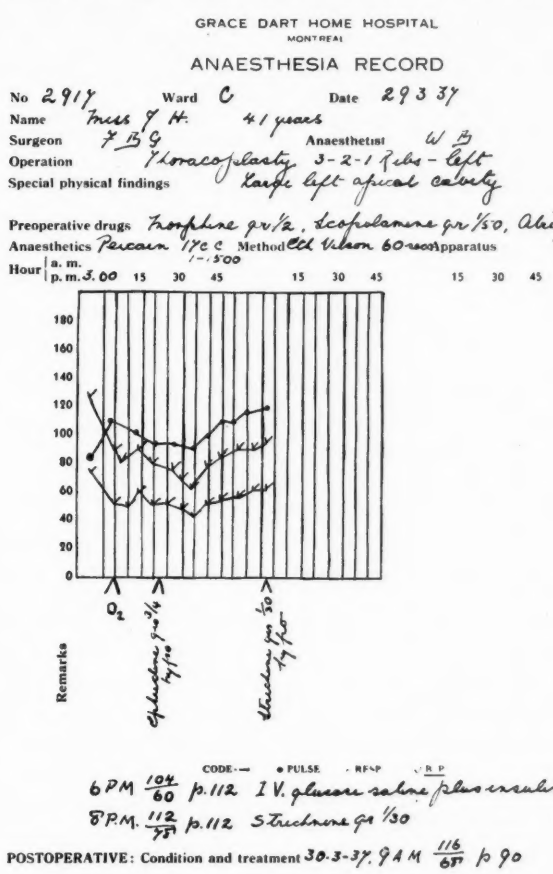

GRACE DART HOME HOSPITAL
MONTREAL
ANAESTHESIA RECORD
No 2917 Ward C Date 29 3 37
Name Mrs J. H. 41 years
Surgeon J. B. G. Anaesthetist W. B.
Operation Thoracoplasty 3-2-1 Ribs - Left
Special physical findings Large left apical cavity
Preoperative drugs Morphine gr 1/2, Scopolamine gr 1/150, Atropine gr 1/150
Anaesthetics Percain 1% c.c. Method Cel Vikon 60 Apparatus
Hour a. m. p. m. 3.00 15 30 45 15 30 45 15 30 45
180
160
140
120
100
80
60
40
20
0
O2
Remarks
CODE — • PULSE · RESP
6 PM 104/60 p. 112 I.V. glucose saline plus insulin
8 P.M. 112/78 p. 112 Strychnine gr 1/30
POSTOPERATIVE: Condition and treatment 30-3-37. 9 A.M. 116/68 p 90

Fig. 1.

hypodermically with the object of alleviating the depression of muscle tonus which spinal anesthesia invariably causes. This is repeated every four hours, for three doses. Also, oxygen and carbon dioxide are administered off and on for several hours after the patient has returned to bed.

The anesthetic chart shown in Fig. 1 is typical and is, therefore, published together with postoperative readings.

The advantages of spinal anesthesia administered by the technique suggested, we believe, may be tabulated as follows:

1. The patient does not lose consciousness, although under the conditions of premedication which we employ sleep is induced and a substantially complete amnesia follows.

2. It is possible to induce the patient to cough, upon instruction. The respiratory effort, particularly if oxygen be administered, is as quiet as may be expected by any form of anesthesia.

3. Owing, presumably chiefly, to the drop in blood pressure and the quiet tranquil respiratory effort, there is less bleeding, we believe, during the course of the operation; at the same time we have not noted any tendency, whatever, toward postoperative hematoma accumulation.

4. In consequence of the quietness of the chest wall and the complete muscle relaxation—of special importance in removal of the transverse processes—and the smaller amount of bleeding, operation can be performed more easily and more expeditiously, therefore, more safely.

5. On return to the ward, cooperation on the part of the patient can be obtained at once, particularly in the sense of deep breathing and purposeful coughing.

6. Although we have made a practice of administering strychnine, and 750 to 1,000 c.c. of 5 per cent glucose in normal saline, to which 10 units of insulin has been added, as a routine in all cases immediately upon return to the ward, this has not been done on account of shock phenomena being exhibited; as a matter of fact, we have not been impressed by shock development in any one case. The highest postoperative pulse rate recorded has been 126 per minute, although most of the cases have remained under 112. Although ephedrine has been administered during the course of operation in approximately half the operations, no other stimulant has been required or used.

7. Patients have been able to commence the ingestion of nourishment within two or three hours of operation, in all cases, and have been able to return to full diet with the lapse of a very much shorter period of relative starvation than in cases in which other anesthetics have been used.

8. The induction period, after the patient arrives in the operating room, is a short and simple procedure.

The objections to spinal anesthesia for thoracoplasty in pulmonary tuberculosis are, we believe, chiefly theoretical:

1. Although the patients do not suffer pain, a few have grunted when traction on the ribs has been made. Using scopolamine and morphine, patients have difficulty in remembering such an incident on the day following operation.

2. There would appear to be a risk of involvement of the phrenic roots; such interference with functioning of the diaphragm has not been noted in any of our cases presumably because the anesthetic is so dilute

when it reaches the midcervical region that motor function is not completely destroyed. Should such an accident develop it would seem probable that serious results might well be avoided by the employment of artificial respiration or intratracheal insufflation.

3. The possibility of a severe circulatory disturbance must be considered. The inevitable drop in blood pressure has, in our experience so far as it minimizes hemorrhage, been useful, and in those cases in which it appeared to be in any way alarming has been promptly controlled by the employment of ephedrine, if necessary administered intravenously.

REFERENCES

1. Shields, H. J.: Spinal Anaesthesia in Thoracic Surgery. Canad. M. A. J. **29**: 528, 1933.
2. Shields, H. J.: Spinal Anesthesia in Thoracic Surgery. Anesth. & Analg. **14**: 193, 1935.
3. Bettman, R. B., and Biesenthal, Max: Extrapleural Thoracoplasty Performed Under Spinal Anaesthesia. Am. J. Surg. **11**: 469, 1931.
4. Newton, Harlan F.: Spinal Anesthesia in Thoracoplastic Operations for Pulmonary Tuberculosis. J. THORACIC SURG. **4**: 414, 1935.
5. Etherington-Wilson, W.: Intrathecal Nerve Root Block. Some Contributions and a New Technique. Proc. Roy. Soc. Med. **27** (pt. I): 323, 1933-4.
6. Etherington-Wilson, W.: Ibid., Brit. J. Anaesth. **9**: 43, 1934; Anesth. & Analg. **14**: 102, 1935.
7. Bourne, Wesley, and O'Shaughnessy, P. E.: The Etherington-Wilson Technique in Intrathecal Segmental Analgesia. Canad. M. A. J. **35**: 536, 1936.
8. Henderson, Yandell: Atelectasis, Massive Collapse and Related Postoperative Conditions, Bull. New York Acad. Med., 2nd Series, **11**: 639, 1935.

DRY AND WET STAGES OF OBSTRUCTIVE ATELECTASIS*

JULIUS KAUNITZ, M.D.
NEW YORK, N. Y.

THE vast amount of literature on the subject of atelectasis since Pasteur's[1] original contribution in 1890 has failed to clarify this interesting subject.

The use of the term atelectasis has been subject to much criticism; many authors have objected that the word explains neither the mechanism of development of the process nor its etiology or sequelae. The usual concept is that of the congenital state, in which the lung is collapsed or airless. This simple condition, however, is rarely found in long-standing cases of bronchial obstruction and subsequent pneumonic consolidation, abscess formation, bronchiectasis, or fibrosis. With the hope of differentiating the conditions brought about by bronchial obstruction from those due to pressure, such as hydro- and pneumothorax, tumors, etc., various authors have suggested the substitution of such terms as active and passive collapse, active and passive atelectasis, and deflation. The term apneumatosis has been presented by Coryllos and Birnbaum,[2] who cover the subject of terminology in great detail.

There does not appear to be any one word which completely describes the condition existing in atelectasis of bronchial obstruction. The term apneumatosis, as shall be shown, is appropriate only at the very beginning of the process, soon after the air has been absorbed by the alveolar capillaries. Atelectasis is a more descriptive term than collapse, as the latter might be misinterpreted as circulatory failure unless qualified as pulmonary or alveolar collapse.

Since the word atelectasis in itself denotes a collapsed state of the lungs it would seem proper to employ it to the exclusion of all others, regardless of all the associated pathologic phenomena, whether pneumonia, abscess, bronchiectasis, or fibrosis. The usual pathogenesis of atelectasis may be reversed, obstructive atelectasis being a sequel to lobar pneumonia. Then also the lung may collapse or shrink because of a chronic sclerotic process, the resulting atelectasis being due neither to compression nor to bronchial obstruction but to a chronic inflammation of the lung.

The characteristic radiographic shadow of obstructive atelectasis is generally considered as representing merely pulmonary condensation. The reason for this is probably to be found in the confusion of this type

*From the Medical Services of Sydenham and Sea View Hospitals.
Received for publication, November 15, 1937.

of pulmonary collapse with that of pneumothorax and congenital atelectasis. Because the mechanism in the development of obstructive atelectasis is different, the radiographic features as well as the clinical and pathologic changes are different.

Ever since the contribution of Pasteur there has been frequent discussion of the cause of massive atelectasis, most authors disagreeing with Pasteur's contention that paralysis of the diaphragm is the cause per se. In this country Coryllos and Birnbaum,[2] who have made notable contributions to this subject through their intensive experimental work, look upon bronchial obstruction as the sole cause. Where the obstruction is due to mucus, its accumulation is said to be aided by such factors as diminution of respiratory movements and coughing because of pain, cachexia, the recumbent position, and narcotics. Elliot and Dingley,[3] in a study of postoperative atelectasis, found in every one of their cases a thick mucopurulent exudate which they traced as far down as the finer bronchioles. They were unable to produce atelectasis in animals by paralyzing the diaphragm, and for this reason concluded that paralysis or immobility of the diaphragm is important only in the presence of a thick bronchial secretion. Hennell[4] reports massive atelectasis in a case of polyneuritis complicated by bronchitis. Here at least two important factors were present: the weakened thoracic musculature and excessive bronchial secretion, mucus accumulating in the bronchi because of the diminished expulsive power. There is an extensive literature dealing with the causes of atelectasis, and for an excellent bibliography on this subject the reader is referred to the works of Coryllos and Birnbaum.

Several important factors may be said to favor the development of postoperative massive collapse: (1) bronchial secretion, (2) diminished cough reflex due to sedatives or anesthetics, (3) weakness of the thoracic musculature, (4) immobility of the thoracic cage and diaphragm due to tight bandaging or pain, and (5) posture favoring the accumulation of secretion in the dependent portions of the lungs. In operations, the presence of an upper respiratory infection is always hazardous because of the likelihood of a complicating atelectasis, followed by a pneumonic process.

A less dramatic form of atelectasis is the so-called patchy variety which occurs in invalids who have been on their backs for long periods. Here the weakened thoracic muscle, the supine position, and the diminished mobility of the posterior chest favor the accumulation of bronchial secretion in the dependent portions of the lung. This patchy atelectasis is probably the forerunner of many cases of hypostatic pneumonia. Hypostatic congestion in the aged, in patients with cardiac disease, and in invalids is generally believed to be responsible for the pneumonia by first diminishing the resistance of the lung to the invading organisms. The infection, however, is bronchogenic and not of vascular origin. This is shown in both gross and microscopic sections, where patchy or lobular

pneumonia is found. It seems reasonable to consider hypostasis of the bronchial secretion at least as important as hypostasis of the blood in the development of hypostatic pneumonia.

The development of obstructive atelectasis depends to a great extent on the cause of the occlusion. Obstruction due to foreign body, neoplasm, inflammatory exudate, aortic aneurysm, etc., may lead to a slow or rapid development of atelectasis. The involvement may be massive or patchy, sterile or infected. A chronic narrowing of the bronchial lumen as by a cicatricial stricture or neoplasm may become suddenly blocked by a blood clot, mucous plug, or other object.

Most authorities are agreed that soon after a bronchus becomes occluded, the pulmonary capillaries begin to absorb the air that normally keeps the alveoli distended. Favored by the normal elastic recoil of the lung tissue, there occurs a general collapse of that portion of the lung supplied by the obstructed bronchus, resulting in a diminution in volume of the lung depending on the bronchus affected. The intrapleural pressure, normally below atmospheric, tends to become lower still because the lung takes up less space within a relatively rigid cage. But inasmuch as the intraabdominal pressure, the contralateral intrapulmonary pressure, and the atmospheric pressure remain relatively unchanged, the diaphragm becomes elevated, the contralateral lung increases in volume, displacing the mediastinal structures toward the affected side, and the thorax becomes depressed on the affected side. This displacement of organs toward the affected side would completely neutralize the tendency of the shrinking lung to diminish the intrapleural pressure, were it not for a limit to which this displacement may occur. The diaphragm is held down by its thoracic and abdominal attachments and can ascend to only a limited degree. The mediastinal structures are restricted in their excursions by the attachments of the pericardium and by pleural reflections to the thoracic wall. The bony thoracic cage is limited in its contraction by the very nature of its structure. Because of these limitations, the effect of massive atelectasis in diminishing the intrapleural pressure is practically never neutralized, in spite of the apparent encroachment of the various organs on the affected lung.

The intrapleural pressure may be markedly depressed in massive atelectasis, some authors reporting a decrease of as much as 40 cm. of water. Elrick[5] reports a case of mediastinal hernia of the opposite lung due to the tremendous negative pressure. How important a part this decreased pressure plays in the further development of the atelectasis is difficult to say. Kaufmann,[6] Loeschke,[7] and others regard it as an explanation for the transudation from the capillaries of some of the blood elements found in the alveolar spaces. Adams, Hrdina, and Dostal[8] explained the blood transudate in the lungs of their experimental animals on the basis of passive congestion. Coryllos and Birnbaum[9] found the alveoli filled with serum and leucocytes in some of their

experimental animals. They regarded these findings, however, as indications of a pneumonic process. These reports are mentioned to indicate that the presence of various liquids in the lungs during atelectasis has been considered by various authors, even though they disagree about the source and character of these liquids.

Mucus is a type of liquid seldom mentioned as present in the lungs, although it is suggested in a description by MacCallum,[10] who speaks of the atelectatic lung as being "moist and sticky." My own attention was first directed to this subject when I found mucus in the syringe after performing a thoracentesis on an atelectatic lung. I have found mucus on several occasions mixed with pus, and once with blood. Many have been dry taps, particularly in tuberculosis where atelectasis has reached the fibrotic stage.

The presence of great quantities of mucus in massive atelectasis is known to all bronchoscopists. Elliot and Dingley, as mentioned, traced thick bronchial secretion to the finer bronchioles. Of their experimental animals, Coryllos and Birnbaum[11] say: "We have never found the bronchi below the obstruction free from secretion." It seems reasonable to suppose that if the trapped bronchial secretion cannot escape, it must ultimately accumulate in the alveoli.

The flooding of the alveoli with liquid, whether bronchial secretion, transudate, or both, should be regarded as part of a natural development in obstructive atelectasis, and not as a complication.

Of course it is not to be expected that the alveolar contents or the pathologic findings will be uniform in experimental or clinical atelectasis. The use of a balloon, ligature, or chemical irritant to obstruct the bronchi probably accounts for the different bacteriologic and pathologic findings. Clinically, the sequelae depend to a great extent upon the type of organisms aspirated. The usual postoperative sequel is pneumonia. Occlusion due to foreign body is more apt to lead to abscess of the lung because of contamination by mouth anaerobes.

In the very chronic forms a denudation of the alveolar epithelium eventually occurs, followed by fibrosis of the alveolar walls and further shrinking of the lung. Another sequel to the obstruction is bronchiectasis, and this may be associated with fibrosis. Considering the different causes of and sequelae to the bronchial obstruction, it is not surprising to find such a contradiction of findings by the various authors. Not until atelectasis is properly grouped as to cause of obstruction, clinical course, and sequelae may we expect any uniformity in its description. This applies to the clinical as well as to the pathologic findings.

I have elsewhere suggested[12] that we divide atelectasis into three stages: (1) early or dry stage, (2) wet stage, and (3) fibrotic stage. The first or dry stage coincides with the absorption of the alveolar gases. Theoretically, the alveolar sacs should be dry, but actually some moisture may enter soon after absorption of gases, and although the lung may not

be absolutely dry, it is dry enough to speak of it as such for purposes of differentiation. In the second or wet stage, the lung is flooded with liquid and gives rise to the symptoms and signs to be described. The fibrotic stage generally follows a persistent bronchial obstruction.

The physical signs are pertinent here only insofar as they indicate the two important stages in the development of obstructive atelectasis.

Soon after the bronchial obstruction in massive atelectasis, the resonance of the affected lung is found to be impaired. The area of cardiac dullness is considerably diminished, with the apical impulse shifted toward the side of the obstruction. The border of liver dullness is found higher and continuous with that of the affected lung. Voice sounds and vocal and tactile fremitus are diminished, and fine crepitant râles are heard such as occur at the margin of lungs or over an azygos lobe. In the upper lobe the breath sounds are tracheal because of the marked deviation of the trachea toward this portion of the lung. These signs frequently lead to a diagnosis of pneumonia. The changes in the pulmonary, cardiac, and liver dullness represent the absorption of alveolar gases followed by shrinking of the lung and displacement of the contiguous organs. The presence of crepitant râles, however, indicates that a certain amount of moisture is already present in the finer bronchioles or alveoli. When bronchial obstruction is due to a foreign body, wheezing sounds, as in asthma, may be heard over the whole chest. This generalized bronchial spasm is probably a reflex manifestation to the foreign body.

In the second or wet stage the size and expansion of the homolateral hemithorax are decreased, and the percussion note is flat. The breath and voice sounds as well as vocal and tactile fremitus are absent below. Tracheal voice and breath sounds may be heard in the upper lobe. Excepting the flattening of the hemithorax and the displacement of the heart and trachea toward the affected side, the physical signs at this stage are identical with those of pleural effusion.

The stage of fibrosis should be mentioned here because of the difficulty of differentiating it from the wet stage. The pulmonary resonance, voice and breath sounds and fremitus, as well as cardiac and tracheal displacement, are the same in both conditions; the tracheal breath sounds appear to be much better conducted in the fibrotic type. Marked conduction of tracheal breath sounds is frequently found in the fibrotic type of atelectasis complicating tuberculosis as a result of cicatricial stricture of a bronchus.

Complications such as pneumonia, pulmonary abscess and bronchiectasis bring in their own train of physical signs.

The x-ray shadows in the very early stage of postoperative atelectasis already show the effect of the diminished size of the lung. The heart and trachea appear to be shifted toward the affected side, and the corresponding leaf of the diaphragm is elevated. The lung appears less

aerated than normal and is frequently mottled (Fig. 1*A*). This is a case of postoperative atelectasis, the obstruction being in the right upper lobe bronchus, and the shift of the heart and trachea toward the upper lobe is quite marked. The diaphragm is elevated, but not to the same extent as in cases where the main bronchus is completely occluded.

The upper lobe appears slightly opaque with dense streaks radiating upward and outward from the hilum. These shadows have an opacity comparable with those cast by the heart and diaphragm or by a pleural effusion, and most probably represent liquid-containing areas of the lung. From this point the process may undergo resolution or continue to the wet stage.

In Fig. 1*B* the same lung is shown two days later, after carbon dioxide and postural therapy. The heart and trachea are replaced toward the midline. The right lung appears better aerated, and, with few excep-

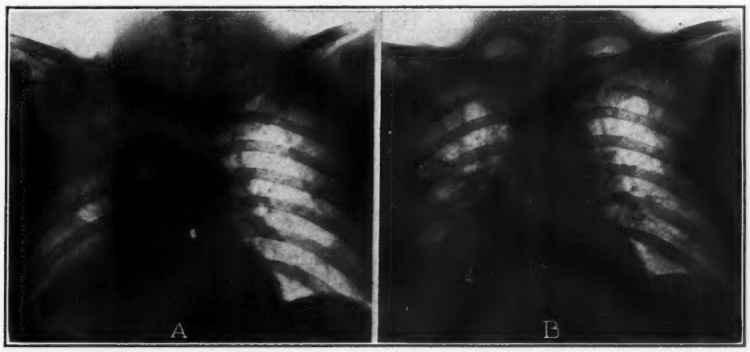


Fig. 1.—Illustrating the first or dry stage of atelectasis, due to postoperative obstruction of the right upper lobe bronchus.

A, The heart, mediastinum, and trachea are dislocated toward the affected lobe. The right lung is poorly illuminated, particularly the upper lobe in which there are patchy dense shadows radiating upward and outward from the hilum. These shadows, which are as dense as those of the heart and liver, may be attributed to early accumulation of liquid. The diaphragm is not elevated to the same extent as when the lower lobe bronchus is also occluded.

B, Same case, after two days of postural drainage and carbon dioxide therapy. The heart and trachea are replaced toward the midline. The right lung now appears better aerated and the dense radiating shadows have practically disappeared, suggesting expulsion of mucus from the bronchus as well as absorption and evaporation of the liquid in the lung.

tions, the dense radiating shadows over the upper lobe have disappeared. These pectorograms indicate that atelectasis may undergo resolution soon after the first stage and never reach the second or wet stage, provided that the obstruction is removed early enough.

In the second stage, after the lung is filled with liquid, we obtain (Fig. 2*A*) a homogeneous shadow so dense that it completely obscures the ribs and merges with shadows cast by the heart, liver, and diaphragm. Excepting for the displacement of the heart and trachea toward the affected side, this picture can be mistaken for pleural effusion.

The coincidence of pleural effusion and atelectasis is not altogether a rarity, particularly in neoplasms of the bronchus or mediastinum. The

case illustrated by Fig. 2*A* is one of atelectasis due to obstruction of the right main bronchus by an endobronchial carcinoma. The dense shadow is due to the presence of liquid in the lung and not in the pleural space. This was proved by the following procedure: a needle connected through a three-way stopcock to a syringe and pneumothorax manometer was introduced into the lung and 10 c.c. of a thin, light, cloudy, mucoid liquid aspirated. The needle was then gradually withdrawn until it entered the pleural space, which was recognized by the sudden fall in pressure to –26, –12 cm. water. A pneumothorax was now induced by injecting 500 c.c. air (Fig. 2*B*). Had any liquid been present in the pleural space, a liquid level characteristic of hydropneumothorax would have formed, the dynamics of which have been explained elsewhere.[13]

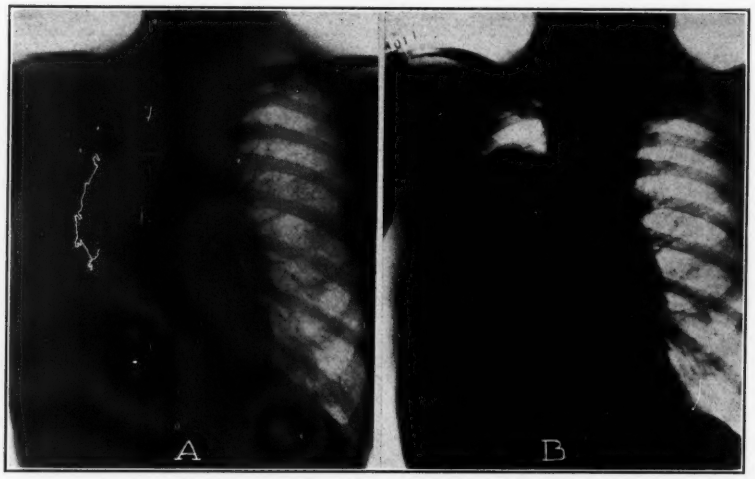


Fig. 2.—Illustrating the second or wet stage of atelectasis, due to old bronchogenic carcinoma of the right main bronchus.

A, The right chest is contracted, and the heart, mediastinum, and trachea are dislocated toward the affected side. The dense homogeneous shadow which resembles pleural effusion is due to liquid in the lung and not in the pleural space. The shadow due to the liquid is comparable in density with that of the heart and liver.

B, Same case as 2*A*. A needle connected to a syringe and pneumothorax manometer was introduced into the lung and 10 c.c. of a seromucoid liquid aspirated. The needle was then withdrawn until it entered a free pleural space, which was recognized by the sudden fall in pressure to -26, -12 cm. water. Then 500 c.c. of air was introduced, creating a pneumothorax shown above the second rib. Absence of a liquid level helps to confirm that the liquid was aspirated from the lung and not from the pleural space.

The result of the induced pneumothorax seen in Fig. 2*B* is an air space above the second rib. Below this the lung as well as the ribs are now better visualized because of the interposition of air between the lung and chest wall. The lung appears mottled at the apex; the heart and trachea have been only slightly shifted toward the midline by the pneumothorax. It appears evident that the dense homogeneous shadow is not due to intrapleural liquid but rather to liquid in the lung proper.

Both the obstruction and reopening of a bronchus can frequently be followed by x-ray studies (Fig. 3). This is a case of postoperative obstructive atelectasis. In Fig. 3*A* the greater part of the right upper

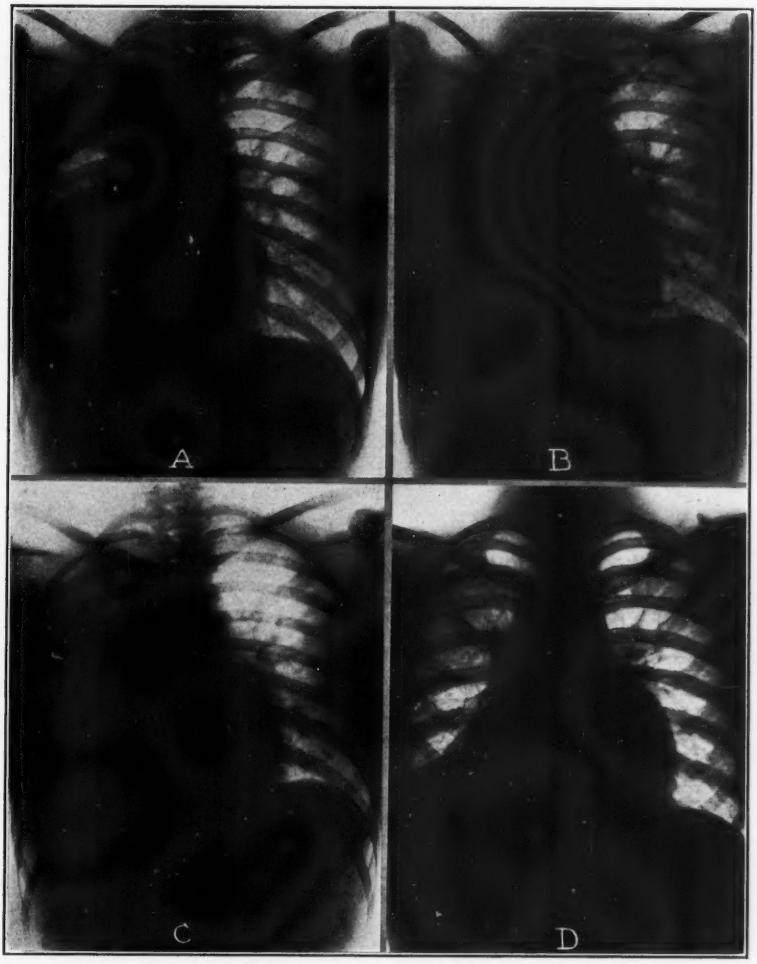


Fig. 3.—Case of postoperative obstructive atelectasis, demonstrating progressive changes following closure and reopening of the bronchi.

A, December 30, 1936. Obstruction of the right upper and middle lobe bronchi. Wet stage of atelectasis in the lower two-thirds of the right upper lobe and dry stage at apex and middle lobe. Heart and trachea are dislocated toward the right. Right diaphragm is elevated.

B, December 31, 1936. Obstruction now includes right lower main bronchus. The entire right lung is in the wet stage of atelectasis. Heart and trachea dislocated to the right.

C, January 2, 1937. Clearing in the upper lobe indicates removal of obstruction in the upper lobe bronchus. The middle and lower lobes remain flooded with liquid.

D, January 11, 1937. Removal of bronchial obstruction on the right side fairly complete. The small triangular shadow to the right of the heart indicates residual atelectasis. The heart, trachea, and diaphragm are now practically in their normal positions.

lobe is in the wet stage while a part of the apex and middle lobe shows the early or dry stage. This may be attributed to a beginning obstruction of the upper and middle lobe bronchi. In Fig. 3*B*, taken the following day, the obstruction has included the lower lobe bronchus, and we now find the entire lung field presenting one dense homogeneous shadow indicating the wet stage. Dislocation of the trachea and heart to the affected side is noted in both roentgenograms. Elevation of the diaphragm can be observed only in Fig. 3*A*, before the lower lobe has been flooded with liquid. Resolution a few days later following removal of the obstruction is seen in Fig. 3*C*. Clearing beginning in the upper lobe is indicative of an early reopening of the upper lobe bronchus. The mediastinal structures remain dislocated toward the atelectatic lung.

Fig. 4.—Azygos lobe atelectasis. The shadow cast by this lobe is only moderately opaque and represents condensation. A small amount of liquid is probably trapped in the alveoli and bronchioles.

In Fig. 3*D*, taken nine days later, a more complete resolution is noticed. The heart and trachea are nearer to their normal location. The triangular area of density to the right of the heart and the slight elevation of the diaphragm suggest some residual atelectasis.

For the purpose of comparison, pectorograms of azygos lobe, pneumothorax, and congenital atelectasis are presented.

The azygos lobe (Fig. 4) is recognized by a moderately dense shadow at the upper part of the right side of the chest near the sternum. Azygos lobes, which are usually described as atelectatic, are frequently mistaken for tuberculous lesions, particularly because of the signs of impaired resonance and râles. It is to be noted that the shadow cast by the azygos lobe in no way compares with the dense shadow of the wet lung,

inasmuch as the latter obscures all the bony structures. The presence of râles in the azygos lobe, however, indicates that some moisture is present.

In Fig. 5 is shown a normal right lung collapsed by pneumothorax. The shadow which it casts is not any denser than that of its normal companion and certainly less opaque than that of the wet lung of obstructive atelectasis. In spite of its collapsed state, this lung expands and contracts, expels mucus from the bronchi and permits evaporation of moisture from its interior. Therefore it should not accumulate liquid as in obstructive atelectasis.

Fig. 5.—Compression atelectasis due to pneumothorax. The right lung, although partly collapsed, does not appear less aerated than its normal companion. There is no evidence of liquid in this type of atelectasis where the bronchus is open, and where secretions may be expelled or may evaporate.

Fig. 6 is a radiograph of a rabbit heart-lung preparation. The right lung is collapsed, the left distended. The heart is held upside down. The purpose of this preparation is to show the relative densities of the atelectatic and distended lungs under the same environmental conditions —unlike pneumothorax, where only the affected lung is surrounded by air. It is to be noticed that the shadow cast by the collapsed lung, although considerably denser than that of its distended companion, is not as dense as the portion of heart containing blood.

Fig. 6.—Rabbit heart-lung preparation, to show the relative densities of the expanded and collapsed lungs under the same environmental conditions, that is, when surrounded by air; this is unlike pneumothorax, where only the affected lung is surrounded by air. The heart is held upside down. It is to be noted that the collapsed lung, although casting a denser shadow than the distended lung, is less radiopaque than the portion of the heart containing blood.

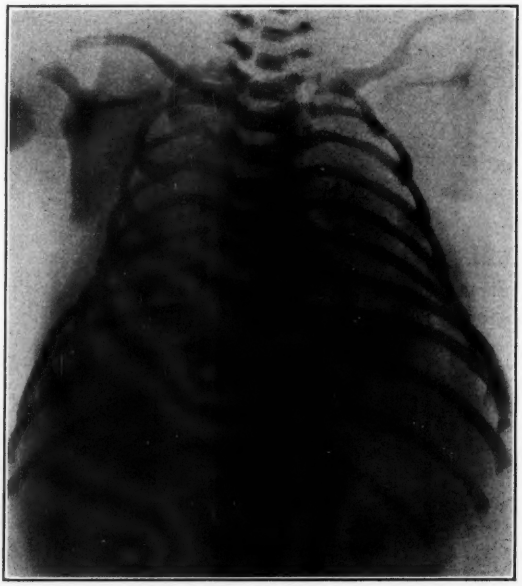


Fig. 7.—Congenital atelectasis. Dry atelectasis, where there is neither liquid in the lung nor gas in the pleura to complicate the picture. The lungs cast a homogeneous radiographic shadow which is faint compared to the shadows cast by the osseous structures.

Probably the simplest type of atelectasis is the congenital form (Fig. 7) where neither pneumothorax nor liquid accumulation have clouded the picture. As would be expected, such lungs cast a rather light shadow as compared with the osseous structures of the thoracic cage. This would correspond to the first or dry stage of atelectasis.

In cases of massive atelectasis of long duration, the lung, as has been mentioned, may become fibrotic, in which case the x-ray shadow may appear dense and homogeneous, exactly as in the wet stage of atelectasis. The coexistence of complicating bronchiectasis may be obscured by the dense shadow in the wet or fibrotic stages unless a successful bronchogram is obtained.

CONCLUSIONS

Obstruction of a bronchus is followed by absorption of the alveolar gases, resulting in diminished intrapulmonary pressure and leading to decreased pulmonary volume and intrapleural pressure. Diminution of intrathoracic pressure is responsible for transudation of blood elements from the capillaries into the alveolar spaces. Bronchial obstruction interferes with expulsion and evaporation of bronchial secretion and alveolar moisture. Because of mechanical obstruction and of changes in the intrathoracic pressure, liquid accumulates in the lung spaces.

Atelectasis may be massive, lobar or lobular, depending on the bronchus occluded. In acute conditions, as in postoperative cases, an entire lung or lobe may be involved. In the aged and in invalids kept for long periods in the decubitus position, stasis of mucus in the smaller bronchi may lead to lobular or patchy atelectasis.

Three arbitrary stages of atelectasis may be said to follow bronchial obstruction: (1) the dry or early stage which occurs with the absorption of alveolar gases; (2) the wet stage, resulting from transudation of plasma and accumulation of bronchial secretion in the alveoli; (3) the fibrotic stage which follows a persistent wet stage and denudation of the alveolar epithelium.

The radiographic shadows of obstructive atelectasis differ in accordance with the stage. In the first stage there is moderate density of the affected lung, the result of pulmonary condensation. As liquid makes its appearance, there is increase in the density, often of a patchy character, until the second or wet stage is reached, when the shadow becomes as dense as those cast by the heart or liver. The shadow may be opaque and homogeneous, resembling pleural effusion, from which it is easily differentiated by the dislocation of the heart, trachea, and mediastinal structures toward the affected lung. The ascent of the diaphragm is recognized in the early stage, but not in the wet or fibrotic stages, where the lung, ribs, heart, and liver are all fused into one dense homogeneous shadow.

REFERENCES

1. Pasteur, W.: Respiratory Paralysis After Diphtheria as a Cause of Pulmonary Complications, With Suggestions as to Treatment, Am. J. M. Sc. **100**: 242, 1890.
2. Coryllos, P. N., and Birnbaum, G. L.: Obstructive Massive Atelectasis, Arch. Surg. **16**: 501, 1928.
3. Elliot, T. R., and Dingley, L. A.: Massive Collapse of Lungs Following Abdominal Operations, Lancet **1**: 1305, 1914.
4. Hennell, H.: Massive Pulmonary Atelectasis: Its Probable Mechanism and Clinical Significance, Arch. Int. Med. **44**: 604, 1929.
5. Elrick, L.: Mediastinal Hernia Following Massive Atelectasis, Am. Rev. Tuberc. **36**: 398, 1937.
6. Kaufmann, Edward: Pathology for Students and Practitioners, Vol. 1, p. 357, Philadelphia, 1929, P. Blakiston's Son & Co.
7. Loeschke, H.: Handbuch d. Spec. Path. Anat. u. Hist. Henke-Lubarsch. **3**: part 1, 559.
8. Adams, W. E., Hrdina, L., and Dostal, L. E.: Vascular Changes in Experimental Atelectasis, J. THORACIC SURG. **4**: 377, 1935.
9. Coryllos, P. N., and Birnbaum, G. L.: Ref. 2, p. 541.
10. MacCallum, W. G.: A Textbook of Pathology, Philadelphia, 1933, p. 412, W. B. Saunders Company.
11. Coryllos, P. N., and Birnbaum, G. L.: Ref. 2, p. 41.
12. Kaunitz, J.: Obstructive Atelectasis: Radiographic Shadows of the Dry and Wet Stages, Bull. Sea View Hosp. **2**: 290, 1937.
13. Kaunitz, J.: Liquid Levels and Other Liquid Surfaces in Pleural Effusions, J. THORACIC SURG. **4**: 300, 1935.

STERILIZATION OF THE AIR IN THE OPERATING ROOM WITH BACTERICIDAL RADIATION*

Comparative Analysis of 132 Individual Stages of Extrapleural Thoracoplasties Performed With Radiation and 110 Stages Performed Without Radiation

Deryl Hart, M.D.
Durham, N. C.

DURING the past fifteen months we have performed 132 individual stages of extrapleural thoracoplasties on 59 patients in a field of air sterilized by means of bactericidal radiant energy. The method was described and the results obtained in the first 18 cases were presented[1] at the 1936 meeting of the American Association for Thoracic Surgery.

The improvement in clinical results brought about by sterilization of the air with bactericidal radiation will be presented under the following headings: (1) mortality, (2) infection, (3) postoperative temperature elevation, (4) duration of elevated temperature after operation, (5) wound healing, (6) general reaction of the patient, (7) changes in the operative procedure.

The various statistics are given in the tables, and for purposes of comparison similar statistics are given for the 110 thoracoplasties (individual stages) performed between 1930 and 1936 without sterilization of the air.

Mortality.—The mortality has been more than cut in half by the elimination of cases of severe wound infection which resulted in death (Table I). There have been three deaths, all of which might have been avoided.

The first patient, a white woman with tracheobronchial tuberculosis, died of respiratory obstruction four days after operation in spite of the use of an oxygen tent and final resort to tracheotomy. This patient should not have been operated upon.

The second patient, a colored woman, stopped breathing during the induction stage of nitrous oxide and oxygen anesthesia. After a period of artificial respiration, her condition seemed satisfactory and it was decided to proceed with the operation. She stopped breathing again after the removal of parts of the second and third ribs. Every effort at resuscitation failed.

---

*From the Department of Surgery, Duke University School of Medicine and Hospital.
Read before the Twentieth Annual Meeting of the American Association for Thoracic Surgery at Saranac Lake, N. Y., May 31, June 1 and 2, 1937.

The third patient, a white woman, had five ribs resected thirteen days after the first stage, at which time the upper three ribs had been removed. The condition of the patient was good throughout the time of the resections, and there was little mobilization of the lung due to fibrosis and inflammatory reaction. The patient went into shock during the closure and died within a few minutes. The operator ascribed the death to the magnitude of the operation carried out at one stage because of his desire to save the patient from a third operative procedure.

Infections.—Before beginning the use of bactericidal radiation all thoracoplasties were drained, since there was an occasional severe infection which necessitated opening the wound widely. In four of these patients death occurred as a result of the infection. Our records show that an organism, usually the hemolytic *Staphylococcus aureus,* was cultured from the wound in 33 per cent of the cases. Probably the only reason that this percentage was not higher was the lack of diligence on the part of the internes in obtaining cultures. Unquestionably, all wounds were contaminated, and many of them drained for several days. The first three patients operated on in a field of bactericidal radiation were drained. Cultures of the drains on removal failed to show any growth, and no drains have been inserted at operation since that time.

There have been five infections (3.8 per cent, Tables I, II and III), only one of which was severe, and this was the only one which occurred in a primary incision (46 per cent of the total operations). This infection was anticipated since there was gross contamination of the wound with infected perspiration as the result of a large rent in the

TABLE I

INFECTION AND MORTALITY STATISTICS. COMPARISON OF RESULTS WITH AND WITHOUT BACTERICIDAL RADIATION

	WITH RADIATION		WITHOUT RADIATION	
	NO.	%	NO.	%
Total number of operations	132	100.0	110	100.0
Infections	5	3.8	36	33.0
Mortality from infection	0	0	4	3.6
Total mortality	3	2.3	6	5.5

Percentage is based on total number of operations and not on total number of patients.

TABLE II

INFECTIONS IN INCISIONS PERFORMED IN A FIELD OF BACTERICIDAL RADIATION. COMPARISON OF RESULTS OBTAINED IN PRIMARY INCISIONS WITH THOSE OBTAINED IN INCISIONS WHICH WERE REOPENED FOR SECOND AND THIRD STAGES

	NO.	NUMBER INFECTED	% INFECTED	CAUSE OF INFECTION
Primary incisions	61	1	1.6	Gross contamination at operation
Incisions reopened	71	4	5.6	Organisms entered subcutaneous tissues along sutures for first stage?

TABLE III

POSTOPERATIVE INFECTIONS WITH BACTERICIDAL RADIATION—SUMMARY OF CASES

CASE NUMBER	TYPE OF OPERATION	INFECTION DIAGNOSED: DAYS AFTER OPERATION	LOCATION OF INFECTION	INFECTION RESULTED FROM	ORGANISMS	SEVERITY OF INFECTION	RESULT
A	Thoracoplasty. Reopened wound for third stage	7	Superficial localized	Bacteria carried in by sutures for preceding stage or by aspiration of hematoma?	*Staphylococcus aureus*	Mild	Recovery
B	Thoracoplasty. Reopened wound for second stage	21	Superficial localized	Bacteria carried in by skin sutures for preceding stage?	*Staphylococcus albus*	Mild	Recovery
C	Thoracoplasty. Reopened wound for second stage	6	Superficial localized	Bacteria carried in by skin sutures for preceding stage?	*Staphylococcus aureus*	Mild	Recovery
D	Thoracoplasty. Reopened wound for second stage	12	Beneath scapula	Bacteria carried in by skin sutures for preceding stage?	*Staphylococcus aureus* (hemolytic)	Moderately severe	Recovery
E	Thoracoplasty first stage	Gross contamination during operation. Opened on seventh day, widely.	Extensive throughout wound	Gross soiling with infected perspiration. Torn glove.	*Staphylococcus aureus*	Severe	Recovery

Total No. operations 132; total No. infections 5; total infections 3.8 per cent; unexplained infections 3 per cent.

operator's glove on a hot day. The other four followed reopening of the wound for the second or third stage (54 per cent of the total operations). In no case was it severe enough to threaten the life of the patient.

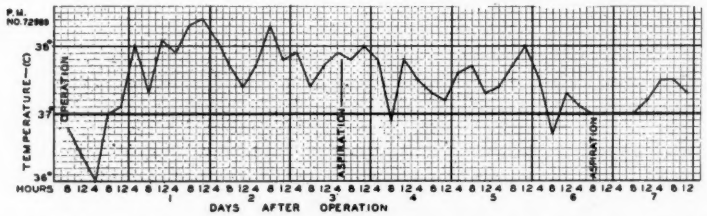


Fig. 1.—No. 72989. Thoracoplasty, incision reopened for third stage. After the first two stages (8 ribs removed) the temperature remained below 38° C. (100.4° F.). After the third stage, bloody fluid, not having the appearance of pus, was aspirated on the fourth and seventh days. Material from the second aspiration showed a positive growth of *Staphylococcus aureus*. Temperature remained low. Infection cleared up with drainage.

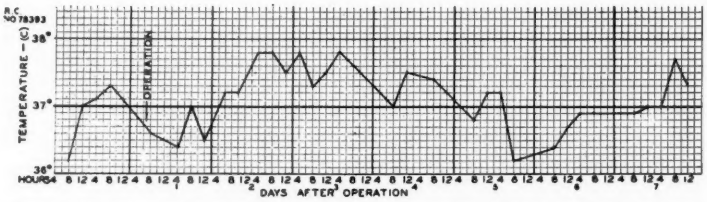


Fig. 2.—No. 78393. Thoracoplasty, incision reopened for second stage. The temperature remained below 37.7° C. (100° F.) after first stage (3 ribs). After the second stage only on four occasions during the twenty-one days before the infection showed up was the temperature recorded above 38° C. (100.4° F.). The organisms, white staphylococci, were harbored about silk sutures in the superficial tissues.

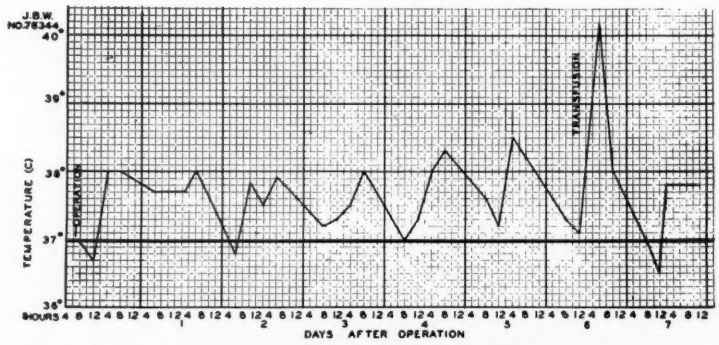


Fig. 3.—No. 78344. Thoracoplasty, incision reopened for second stage. After the first stage (upper 4 ribs) the temperature rose to 38.6° C. (101.5° F.). This was only 0.6° C. above the preoperative level, and it was down to normal within four days. After the second stage (next 4 ribs) an area of superficial infection (*Staphylococcus aureus*) extending about two inches along the incision was found on the sixth day. Healing was rapid following drainage.

In three wounds the infection was superficial, while in the fourth it was beneath the scapula. The temperature curve for the first week and a summary of each infected case is given. (Table III and Figs. 1-5.)

It is our impression that in the four cases in which infection followed reopening of the wound, the organisms gained entrance to the subcutaneous tissues along the skin sutures for the preceding stage. Recently a number of incisions, which would have to be reopened, have been closed with a subcuticular suture of wire. There has been excellent healing, and, to date, no infection has occurred following reopening a wound closed in this manner.

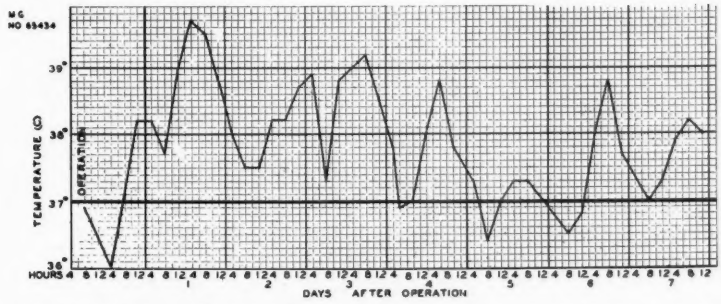


Fig. 4.—No. 65434. Thoracoplasty, incision reopened for second stage. After the first stage (3 upper ribs) the four-hour temperature record showed only four elevations above 38° C. (100.4° F.) and remained normal after three days. Twelve days after the second stage (next 3 ribs) a deep-seated infected hematoma (*Staphylococcus aureus*) was discovered beneath the scapula. The infection subsided with drainage.

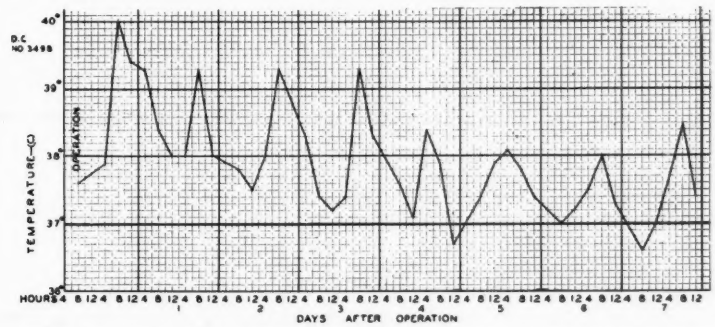


Fig. 5.—No. 3498. Thoracoplasty—first stage. Grossly contaminated with infected perspiration. During the first stage (4 upper ribs) performed on a hot day, there was gross contamination of the wound with infected perspiration as the result of a torn glove. The wound was thoroughly washed out with normal salt solution and had to be opened widely seven days after operation. The infection (hemolytic *Staphylococcus aureus*) gradually subsided, but it was nine weeks before healing was complete so that the second stage could be performed.

Postoperative Temperature Elevation.—Sterilization of the air was started during the winter. There was an immediate drop in the average postoperative temperature elevation (Table IV) so that more than two-thirds of the patients did not have more than two recorded elevations above 38° C. following operation, as compared to one-third of the patients without radiation, who fell in this group. With the warmer months (May 15 to October 15) the patients began to run a higher

temperature, but still much lower than before the introduction of the radiation (Table V). The higher temperature during the summer we ascribe partly to the heat but more to the possible contamination of the wound with infected perspiration (from the skin of either the patient

TABLE IV

TEMPERATURE ELEVATION. COMPARISON OF RESULTS FOLLOWING OPERATIONS PERFORMED WITH AND WITHOUT BACTERICIDAL RADIATION

BEFORE OPERATION					AFTER OPERATION			
WITH RADIATION		WITHOUT RADIATION		TEMPERATURE	WITH RADIATION		WITHOUT RADIATION	
NO.	%	NO.	%		NO.	%	NO.	%
114	86.4	97	88.2	37°-37.5°	30	67	2	32
15	11.4	12	10.8	37°-38°	58		33	
3	2.2	1	1.0	37°-39°	38	33	58	68
0	0	0	0	37°-40°	5*		16	
				37°-41°	1*		1	

*In Tables IV-VI the temperatures above 39° are accounted for in five of the six cases as follows: infection, two cases; extension opposite lung, two cases; temperature of 39° before operation, one case.

or some member of the operating team). During the cooler part of the year 72 per cent of the operations were followed by a temperature falling in the 37°-38° C. (98.6°-100.4° F.) group, while in the hotter months only 51 per cent of the operations were followed by a comparably low temperature (Table V).

TABLE V

POSTOPERATIVE TEMPERATURE ELEVATION WITH RADIATION VARIATIONS DEPENDENT ON THE TIME OF YEAR

TEMPERATURE	COOL (OCT. 15 TO MAY 15)		WARM (MAY 15 TO OCT. 15)	
	NO.	%	NO.	%
37°-37.5°	27	72	3	51
37°-38°	43		15	
37°-39°	24	28	14	49
37°-40°	3		2	
37°-41°	0		1	
Total	97	100	35	100

The temperature elevation also varied with the stage of the thoracoplasty. The first stage was followed by a temperature in the 37°-38° C. (98.4°-100.4° F.) group in only 53 per cent of the cases (Table VI). The second and third stages were followed by this low temperature in 80 per cent of the cases (Table VI) even though these operations have been performed by reopening the old incision at two- to three-week intervals and even though four of the five infections have occurred in these stages (Table II). It is thought that the higher temperature following the first stage frequently results from the disturbance of the tuberculous process.

TABLE VI

TEMPERATURE ELEVATION WITH RADIATION

COMPARISON OF THE POSTOPERATIVE TEMPERATURE ELEVATION FOR DIFFERENT STAGES

BEFORE OPERATION			AFTER OPERATION							
		TEMPERATURE	FIRST STAGE 59 OPERATIONS		SECOND STAGE 48 OPERATIONS		THIRD STAGE 23 OPERATIONS		ANTERIOR 2 OPERATIONS	
NO.	%		NO.	%	NO.	%	NO.	%	NO.	%
114	86.4	37°-37.5°	9	53	15	80	5	80	1	50
15	11.4	37°-38°	22		23		13		0	
3	2.2	37°-39°	24	47	9	20	5	20	0	50
0	0	37°-40°	3		1		0		1	
0	0	37°-41°	1		0		0		0	

Duration of Temperature Elevation.—The duration of the elevated temperature following operation has been greatly shortened by sterilizing the air (Table VII). Where the air has been sterilized the duration

TABLE VII

DURATION OF ELEVATED TEMPERATURE AFTER OPERATION

COMPARISON OF RESULTS WITH AND WITHOUT BACTERICIDAL RADIATION

DAYS	WITH RADIATION		WITHOUT RADIATION	
	NO.	%	NO.	%
0	35	80	2	22
1	3		0	
2	27		1	
3	16		11	
4	23		10	
5- 7	16	20	35	78
8-14	7*		39	
15-21	3*		6	
21+	2*		6	

*The temperature elevations of more than seven days are accounted for as follows:

1-2 weeks:	Unexplained	3
	Extension of tuberculous process	1
	Tuberculous empyema with secondary infection	2
	Infected wound	1
2-3 weeks:	Infected wound	2
	Extension tuberculous process	1
3 + weeks:	Infected wound	1
	Extension tuberculous process	1

has been further affected by the season. During the warmer part of the year the percentage of patients running a temperature for more than four days after operation has been more than three times as great as during the cooler months (Table VIII). The percentage of patients running a temperature elevation for four days or less was practically the same (78-83 per cent) for all stages (Table IX).

Wound Healing.—With radiation the wound healing has been more rapid and with less reaction when reopened for the second and third stages than in those cases in which radiation was not used (Table X).

TABLE VIII

DURATION OF POSTOPERATIVE TEMPERATURE ELEVATION WITH RADIATION. VARIATIONS DEPENDENT ON THE TIME OF YEAR

	COOL (OCT. 15 TO MAY 15)		WARM (MAY 15 TO OCT. 15)	
TOTAL OPERATIONS	97		35	
DAYS	NO.	%	NO.	%
0	31		4	
1	2		1	
2	20	87	7	57
3	14		2	
4	17		6	
5-7	7		9	
1-2 weeks	4*	13	3*	43
2-3 weeks	1*		2*	
3+ weeks	1*		1*	

*See footnote Table VII.

TABLE IX

DURATION OF POSTOPERATIVE TEMPERATURE ELEVATION WITH RADIATION. COMPARISON OF DIFFERENT STAGES

DAYS	STAGE I		STAGE II		STAGE III		ANTERIOR	
	NO.	%	NO.	%	NO.	%	NO.	%
0	14		15		5		1	
1	2		1		0		0	
2	10	78	10	80	7	83	0	50
3	9		4		3		0	
4	11		8		4		0	
5- 7	8		5		3		0	
8-14	2	22	4	20	0	17	1	50
15-21	2		1		0		0	
21+	1		0		1		0	

TABLE X

TIME REQUIRED FOR HEALING. COMPARISON OF RESULTS WITH AND WITHOUT BACTERICIDAL RADIATION

	WITH RADIATION	WITHOUT RADIATION
	DAYS	DAYS
Uninfected cases		
Average	8	21
Shortest	5	11
Longest	66	44
Infected cases		
Average	44	34
Shortest	20	16
Longest	83	73

Wounds have also been reopened at an earlier date than in those cases in which the radiation was not used. In certain recent cases where the closure has been made with wire instead of catgut there has been still less reaction noted when the wound was reopened (Figs. 6 and 7).

General Reaction of the Patient.—The postoperative condition of the patient has been greatly improved. The internes' postoperative notes record 95.5 per cent of the patients as being in good condition (this group includes some patients with low blood pressure who responded well to transfusion), 1.5 per cent as being in fair condition, and 3 per cent as being in poor condition. The last two groups were composed of three

Fig. 6. Fig. 7.

Fig. 6.—Photograph showing the scar of an extrapleural thoracoplasty closed with a subcuticular wire. At the second stage, fourteen days after the first stage, the wound was reopened without excision of the scar and extended at the lower end. The wire was removed, four ribs were resected, and the incision was again closed with continuous sutures of wire. Photograph taken ten days after the second operation.

Fig. 7.—Photograph of the patient shown in Fig. 6 taken ten days after the third stage, which was performed fifteen days following the second stage. The scar was not excised at either the second or third stage. At the third stage the wound was closed with catgut for the muscle and subcutaneous tissue and with silk for the skin. Wire was not used since no further operative procedure was anticipated. The oblique part of the scar is the result of three incisions performed at intervals of fourteen and fifteen days without excision of the scar.

patients who showed an extension of the tuberculous process; two, an infection in the wound; and one, a respiratory obstruction from tracheobronchial tuberculosis. In practically all cases the postoperative pain has been less, and convalescence has been more rapid than in the cases in which radiation was not used.

Changes in the Operative Procedure.—The interval between stages has been shortened to thirteen to sixteen days in most of the recent cases. The later stages are performed by reopening and extending the former

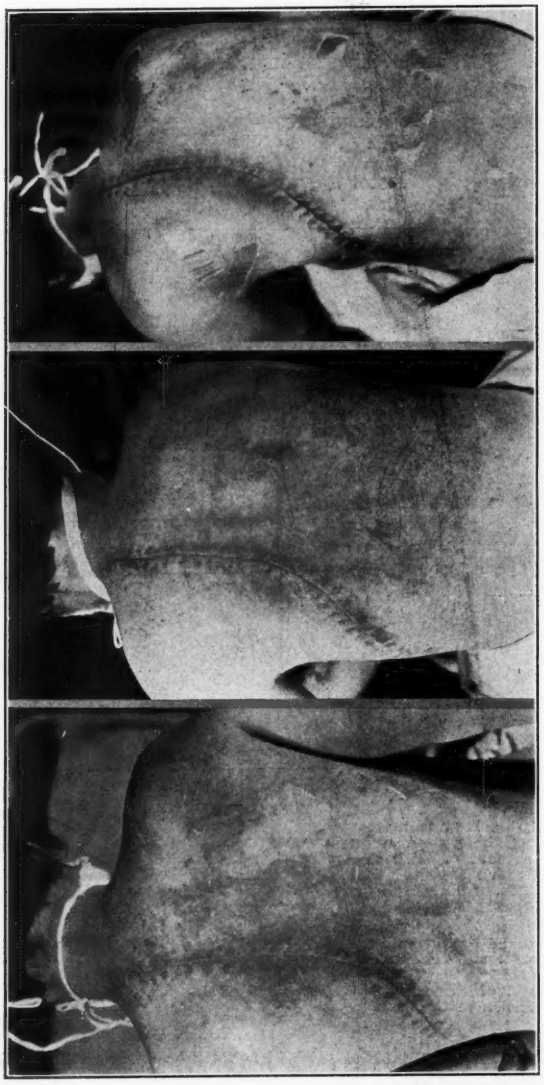


Fig. 8.

Fig. 9.

Fig. 10.

Fig. 8.—Photograph taken twelve days after the first stage. Muscle and subcutaneous tissue closed with catgut. Skin closed with silk.

Fig. 9.—Photograph of patient shown in Fig. 8 taken thirteen days after the second stage which was performed twenty-one days after the first stage. The scar was not excised. Wound closed with buried sutures of catgut and skin sutures of silk.

Fig. 10.—Photograph of the patient shown in Figs. 8 and 9 taken sixteen days after the third stage which was performed thirteen days after the second stage. Scar not excised. Wound closed with buried sutures of catgut and skin sutures of silk. The flap of soft parts was retracted medially for removal of the lower ribs. When the incision is reopened in this manner, there is less bleeding, fewer clamps are required, and without two incisions coming together or into close proximity there is less danger of sloughing as a result of interference with the blood supply.

incision rather than by making a new incision (Figs. 8, 9, and 10). The upper flap, including the scapula, is retracted laterally to remove the upper ribs, while the lower flap is retracted medially for removal of the lower ribs.

Conclusions.—It is our impression that every operative incision is contaminated with pathogenic bacteria. With a small area of exposure, avoidance of trauma and strangulation of tissue, meticulous hemostasis, and closure with silk, there is little possibility of the bacteria gaining a foothold. The risk of infection, however, is much greater in an operation such as a thoracoplasty, where such ideal conditions are not possible at all times. Contamination of the wound from the air can be eliminated almost completely by means of bactericidal radiation. With this elimination of contamination with air borne pathogenic bacteria, unexplained infections in primary incisions have been greatly reduced if not eliminated.

The infections in the wounds where a recent incision has been reopened have been mild and localized. It is our impression that in these cases the organisms gain access to the subcutaneous tissues along the skin sutures for the preceding operation. These infections can probably be reduced by closing the preceding operative incisions with subcuticular sutures of wire instead of skin sutures.

Sterilization of the polluted air has closed the greatest remaining source of contamination of clean operative wounds. Contamination along the skin sutures and from infected perspiration must be carefully guarded against, particularly in those cases where the wound is to be reopened.

REFERENCE

1. Hart, Deryl: Sterilization of the Air in the Operating Room by Special Bactericidal Radiant Energy, J. THORACIC SURG. 6: 45, 1936.

DISCUSSION

DR. DAVID T. SMITH, Durham, N. C.—It has been my pleasure and privilege to watch Dr. Hart's work from the beginning. I saw most of his early cases, both before and after each stage of the thoracoplasty; I also saw the patients before and after he introduced his lamp. The difference in the appearance of the patients is more striking than can be shown by the slides. The patients do not have so much shock, and the thing that amazed me was that they wanted to eat. As a medical man, I am always pleased when a patient wants to eat. I was amazed, also, that pathogenic bacteria should be found in such numbers in the air. I would have been convinced that these were pathogenic bacteria had it not been for the experience of some of my assistants with the staphylococci that were recovered from the air in the operating room. They will make very powerful exotoxin which will kill a rabbit in two minutes; and only with such evidence as that was I firmly convinced that these were important organisms.

There is no doubt in my mind of the result of these thousands of cultures that Dr. Hart made in the last six years. When he exposes plates early in the morning when the operating room is empty, there are practically no bacteria in the air. As soon as the nurses and surgeons come into the operating room, pathogenic organisms are thrown off into the air, more probably from breathing than from rubbing off the skin. If any of you wish to make a test, simply take a blood agar plate, and after you are ready for operation, talk through a gauze mask on the plate; I think you will be surprised at the result.

THE SILVER LINEATION ON THE SURFACE OF THE PULMONIC ALVEOLAR WALLS OF THE MATURE CAT, PRODUCED BY APPLYING WEAK SILVER NITRATE SOLUTION AND EXPOSING TO SUNRAYS OR PHOTOGRAPHIC DEVELOPER*

CHARLES C. MACKLIN, M.D., PH.D.
LONDON, CANADA

IT HAS long been known that a network of fine dark lines exists on the walls of alveoli of lungs which, while fresh, have been briefly distended with weak silver nitrate solution, washed out with distilled water, fixed at once, preferably by injection into the bronchial tree, and acted upon by direct sunlight or photographic developer. The literature on the subject is replete with illustrations of the pattern which these lines form: for instance in Koelliker's Handbuch[1] (1899) we find three *en face* views. Much attention has been given to their interpretation. From analogy with similarly produced silver lines along the boundaries of pavement epithelial cells, and of endothelial cells in blood and lymph vessels, it was early surmised that the lines on the alveolar walls marked the borders of the epithelial cells, of entodermal origin, which were thought to cover these walls. This view was strengthened by the observation that the epithelial cells of the bronchioles, with which the surface cells of the alveoli were presumed to be united in a continuous layer, were each encircled by a silvered band, and that the sum total of these bands, when seen in surface view, appeared as a fairly regular mosaic pattern. But the silver-line network on the alveolar wall stands in sharp contrast with that of ordinary epithelium or endothelium in that the meshes show a striking inequality in size; indeed most of the wall of the mature definitive mammalian alveolus is occupied by curious large and irregular areas in which often no nuclei can be demonstrated, with borders pencilled by the silver deposit.

The point of the matter was, and still is, the interpretation of these large enclosures (which I shall speak of as "bare areas"). They were regarded by many as extremely thin and wide cells, analogous to the "respiratory epithelium" of lower forms, such as frogs and turtles, and derived, of course, from the continuous epithelial layer universally ad-

*From the University of Western Ontario, London, Canada.

Presented at the general opening session of the American Association of Anatomists at its Fifty-third Annual Meeting, at the University of Toronto, Toronto, Canada, March 25, 1937.

Received for publication, July 7, 1937.

mitted to line the alveolar walls in the embryo. Where no nucleus was discerned, they were spoken of as "non-nucleated plates" or "squames" by some workers, to whom they appeared as the end result of a high degree of specialization of the original entodermal epithelial cells. Others thought that the bare areas were thin extensions of nucleated epithelial cell bodies, situated in the capillary meshes, and even denied the existence of nonnucleated squames. These hypothetical diaphanous films or flanges were, it is true, invisible in ordinary preparations, but their presence was not to be ruled out on that account alone. So held their votaries who, walking in this matter by faith, not by sight, thought of them as the product of a further differentiation of the cell extensions in amphibia and reptiles. This group viewed the alveolar wall as completely covered by epithelium, so broadening the meaning of the word to include these ordinarily invisible plaques. But more recently a school of thought has arisen which looks upon the bare areas as nonepithelial. Its adherents refuse to credit what their eyes cannot see, and, therefore, assume that the wall here is surfaced only by a connective tissue fabric. Among the bare areas on definitive walls, both of these schools recognize scattered cells occurring singly or in small groups, which are apparently the modified survivors of the original epithelium. Clara (1936)[2] speaks of these cells as "epicytes." Finally, a group of extremists has arisen who deny outright the occurrence of epithelium upon the wall, which they describe as completely "nude." The alveoli thus become, in effect, physiologic interstitial emphysematous spaces. The pros and cons of this epithelium question are set forth in the report of a Round Table Conference on "Pulmonic Alveolar Epithelium" which was held under the auspices of the American Association of Anatomists at Durham, N. C., on April 11, 1936.[3]

Thus, although much has been said and written upon the subject of the nature of the surface of the mature mammalian pulmonic alveolar wall, the evidence has so far not been sufficiently definite to win investigators over to a uniform view. Further study of the problem is obviously necessary. Therefore, since I feel that alveolar silver lineation may have a bearing on the problem, I have ventured to make the following brief report of my recent investigations in this field.

MATERIAL AND METHODS

Typical mammalian lungs from healthy, mature, domestic cats were used. The animals were anesthetized with ether, strapped to a board, and bled thoroughly by cutting the great abdominal vessels. Upon cessation of the heart beat, the chest was carefully opened. The fresh collapsed and almost completely exsanguinated lungs were then treated as follows: The trachea was cannulated and a fresh solution of 0.2 per cent silver nitrate in distilled water was allowed to run in and distend the lungs for a period of one minute. It was immediately drained out,

and the lungs were at once washed out in the same way with two changes of distilled water, emptied and filled with 10 per cent aqueous neutral formalin. The trachea was then clamped and the lungs immersed in 10 per cent formalin, in which they remained for twenty-four hours or more. In one case the operations were done with the lungs in situ, and here the degree of distention was approximately that of full expiration or slightly less. In others the lungs were removed before the fillings, and the degree of distention was approximately that of full inspiration. A solution of silver citrate made according to the formula of R. D. Bensley,[4] and used in place of the silver nitrate, gave excellent results.

When the material had become quite stiff, blocks were taken from different lobes. Some were frozen and cut at about 50μ. Others, after immersion in dioxane, were embedded in paraffin and cut at 3μ, 10μ, and 25μ. Many of the frozen sections were flattened and affixed to the slides before being stained. These were particularly useful, because in them selected areas of alveolar wall, lying flat in the optical plane, could be conveniently explored, even with the oil immersion lens. Others were prepared in the open, or uncollapsed, condition. In cases where the plane of section was parallel to the axis of a bronchiole, the lineation on the junctional region between bronchiole and alveolar duct or alveolus could be surveyed as a continuous whole. It is, of course, essential to study the surfaces of the alveoli and air canals by viewing them *en face*, and this is impossible in ordinary thin sections.

The lines of silver deposit were darkened by exposure of the sections to direct sunlight or to weak photographic developer. As an alternative, it was found possible to darken the silver lines by flooding the interior of the lungs with 1/10 strength hydroquinone solution after rinsing out the excess silver solution and before fixation.

Many of the sections were cleared and mounted without staining. In these the lines stand out with great distinctness. In order to relate the lines to the neighboring tissues, various staining techniques were employed, including Mayer's paracarmine, Masson's polychrome stains, Mallory's connective tissue stain, Weigert's elastic tissue stain, modified Bielschowsky and others. These stained sections displayed the relation of the silver lines to the epicytes, the nuclei of mural cells, the ground membrane or substance, the capillary surfaces, and the various fibers, elastic, collagenous and reticulum. The stereoscopic microscope was found very useful in exploring the thick open sections; and by diligent focussing and close study of the parts, it was possible to ascertain the position and interrelations of the lines with a fair degree of accuracy. Such studies are time-consuming, of course: often an entire evening would be devoted to intensive microscopic work on a relatively small region of lung. On account of the difficulty of getting any considerable area of alveolar wall into the optical plane, and also because of interference by other parts of the wall, it was not possible to obtain ideal

photomicrographs; but some fairly good fields were recorded thus in spite of the obstacles. In interpreting this network of silver lines we must make allowance for the fact that the usual alveolar wall is really a partition between two alveolar spaces, and thus has two surfaces which are very close together; and it follows that the networks of these two surfaces, when seen in face views, are superposed. This optical intermingling of the two networks is particularly close in the flattened frozen sections. However, by appropriate stereoscopic observation and the use of high powers, this confusion is largely resolved in visual work, but cannot be eliminated altogether from the photographs. In places where alveoli abut upon the sheaths of blood vessels or bronchi, or the pleura (Fig. 1), instead of upon a neighboring alveolus, there is only one layer of silver lines. The thin paraffin sections were very valuable in supple-

Fig. 1.—Photomicrograph made from a flattened frozen section with Zeiss 4 mm. apochromatic objective and Zeiss K7X Mobimi ocular, stained with Masson's polychrome hemalum-phloxin-saffron mixture (hydroquinone development and formalin fixation). The section was cut obliquely, and shows the base of an alveolus abutting upon the pleura. Thus there is only one layer of silvered lines. The outer dark zone, in which no detail can be seen, is the flattened side wall of the alveolus. The floor or base is not all in the optical plane, and so all of it is not in focus. It is possible to see the silver lines around the epicytes and the bare areas. The epicytes (see text) are more numerous in proportion to the bare areas than they are on the definitive walls. They are of fairly uniform size, occur singly or in groups of two, three or even more on bases like this abutting upon pleura (or upon the sheaths of bloodvessels or air-tubes). The outlines approach the oval or circular, but often show one or more angles. When in focus, a nucleus can be discerned in each. Each epicyte outline is connected with the silver lines around other epicytes or bare areas. Many of the epicytes here are out of focus. The bare areas are the larger enclosures and have no surface nuclei (for their description, see text). It is, of course, impossible to show in a small photograph the entire pattern of the silver-line network on the wall, for this requires focussing at different levels. Section S63-48R-2-3.

menting the spread alveolar wall preparations in that they gave views of the ends of the lines in relation to neighboring parts, such as the epicytes, and supplied details of all elements of the wall.

Description of the Lines.—The best beginning of this study is made with a thick "open" frozen section of lung substance, cleared after hav-

ing been developed, but not stained. It has been taken preferably from the peripheral region of the lung, where no great bronchi, blood vessels, or masses of connective tissue obstruct the view. The stereoscopic picture is very striking. At first it seems a hopeless tangle, as of fine slightly kinked wires, but careful scrutiny soon brings order out of apparent chaos, and we note that the network encloses definite spaces, the alveoli; and we can also see that these are but outpouchings of the alveolar sacs, ducts, and respiratory bronchioles. Here and there a densely stained fine-meshed tubular network is recognized as a section of the lining of a bronchus or bronchiolus. The blood vessels and connective tissue formations are not prominent in such preparations. The pleura, if it occurs, is easily identified.

On examining the alveolar walls with a higher power we see at once that there are two sizes of mesh which, although belonging to the same system, nevertheless have very outstanding differences. There are (a) large, irregular meshes which occupy by far the greater part of the surface area, and are surrounded by fine moderately crooked silver lines. These are the aforementioned "bare areas." Then there are (b) the conspicuous small ovoid or simple angulated enclosures, of fairly uniform size, marked by more distinct silver lines, with contained substance typically light golden brown in color in the cleared condition. These are the epicytes. They always contain a nucleus, and have obvious substance, in contrast to the bare areas, which appear to be only plots bounded by the silver lines. The epicytes, in these cleared preparations, seem to be caught in the wire entanglement. They usually occur singly, but may be in small groups. Their numbers depend largely on the type of wall under consideration. On the two faces of the partition of the definitive alveolar wall the epicytes are relatively scarce, almost all of the wall being occupied by the bare areas. But if we are dealing with an alveolar base which rests upon the sheath of an artery, vein, or airway (bronchus or bronchiolus) or against the pleura (Fig. 1), then the number of epicytes is greater. In such cases there are often groups of two, three or more, and the space occupied by the bare areas is relatively less. Here the capillary net is less rich (Miller[5]). Where the bare areas are greatest, we find the richest capillary network; this is significant. Cells which seem to be intermediate in type between the definitive epicytes and the cells of the continuous bronchiolar epithelium are found in considerable numbers in the alveoli of more embryonic type, which communicate directly with the respiratory bronchioles.

In these cleared thick sections it is possible to study the network in different parts of the lumen of the bronchial "tree." This tree, as Macklin[6] (1929) has pointed out, may be conveniently considered as having two parts, (1) a main conducting division, which has smooth walls, and (2) a peripheral respiratory division, whose relatively short and fine-calibered tubes are characterized by sacculations—the alveoli and

alveolar sacs—on their thin walls. The respiratory division consists of a very large number of units, the primary pulmonic lobules, and each of these has as its stem the respiratory bronchiole, in which the sacculations are of a primitive nature—small, at first confined to one side of the tube, and also at first lined with a cuboidal type of epithelium. From each stem a much-branched system of sacculated ducts arises. These sacculated tubes are really specialized bronchioles, but they go, in common parlance today, following the usage of Miller and others, as "respiratory bronchioles," "alveolar ducts," and "alveolar sacs," the last-mentioned being blind, save for the pores (Macklin, 1934,[7] 1935,[8] 1936[9]). So rich is the wall of the alveolar duct system in perforations leading into similar tubes and sacs that it looks like a sieve when spread out (Macklin, 1929[10]). These rounded openings on the walls of the respiratory division are formed by the myoelastic sphincters—a part of the bronchial musculature (Macklin, 1929[6]). This part of the lung is readily dilatable, opening and closing something like a bellows as the air flows and ebbs through it with respiratory movements. A summary of our knowledge of this terminal and complicated subdivision of the lung, together with a history of the scholastic evolution of these parts, is to be found in the recent excellent treatise of William Snow Miller (1937[5]). It is necessary to have a picture of these airway terminals in mind if we are to understand the topography of the silver-line system being dealt with here.

In favorable sections it is possible to obtain views of the silver lines in transitional regions between the smooth-walled bronchioles and the sacculated, and here the network is especially worthy of note. In the smooth-walled bronchioles the meshes are small and fairly uniform in size and shape. Their heavy pencilling with silver is characteristic of the continuous simple epithelium which makes up the surface of the main conducting part of the airway. In the junctional region ushering in the respiratory part of the air duct system we note the occurrence, here and there, of somewhat larger meshes, with clearer and lighter interior; and where the epithelium of the respiratory bronchiole passes over upon the surface of the communicating alveoli there is a marked change. The remarkable thing here is a widening and clarification of many of the meshes. As these larger meshes increase in size and number, the epithelial cells in the smaller meshes become relatively reduced in numbers. From a continuous membrane they break up into rows or patches, and finally into small groups, and, at the end, that is on the definitive alveolar walls, they occur as single isolated cells, which are then known as epicytes (Clara, 1936[2]). The impression is that the epicytes are scattered, as we pass from the bronchioles to the alveoli. The epicytes retain about the same size as the regular epithelial cells of the respiratory bronchioles. They tend to be larger than those of the smooth-walled bronchioles. It is in the immediate outpocketings and beginnings

of the branches of the respiratory bronchioles that this "scattering" occurs, and it is due, of course, to the gradual ascendancy upon the surface, of the large meshes which, on definitive walls, are known as the "bare areas," and there make up almost all of the alveolar wall surface. In the transition from the smooth-walled bronchioles to the respiratory bronchioles the regular epithelium is prolonged upon the narrowing continuations of the wall between the outpouchings. These openings become larger and more closely set as we pass peripherally. The respiratory bronchiole soon leads off into a system of alveolar ducts, and the epithelium mosaic, characteristic of the respiratory bronchioles, continues to be prolonged into the beginnings of these tubes as still narrower segments of the actual wall. As we study the borders of these extensions we see that the silver line network passes suddenly and directly over into the wide-meshed surface network of the alveolar walls, with epicytes caught in the smaller meshes.

Continuity of the Network.—Thus it is evident that the silver-line network of the bronchioles passes directly over into that of the terminal tubes and alveolar outpouchings. There is doubtless a continuous meshwork of silver lines from the larynx to the alveoli. This is to be expected from the development of these parts, which arise as an entodermal outpouching of the cranial end of the primitive digestive tract. The silver lines are fundamentally related to the epithelium, and there is in the embryo a continuous layer of epithelium in the alveolar walls which are the blind ends of the primitive bronchioles. Thus the silver lines on the alveolar walls themselves are a part of the original epithelial layer which covered these walls in the embryo, and which still covers them in lower forms, as the frog. The interesting thing is the change which has gone on in the epithelium during the formation of the bare areas (and consequently also in the disposition of the argyrophilic material which surrounds each cell), and the ultimate nature of the surface represented by these bare areas. Before discussing these problems, we shall take up certain additional points in connection with the lines.

The physical basis of the silver lines is discussed in the literature, but we do not yet have the final answer. Obviously, we must be dealing with material which has a special affinity for silver, for when a relatively weak solution of silver salt is brought into contact with this for only a brief time, there is so much silver taken up that it appears dense brown or even black after development. It is upon this argyrophilic property that good visibility of this material depends. It has been thought to be a sort of cement substance which occurs between the epithelial cells. It must be readily adaptable to lengthening associated with the increase in the surface area of the cells which it surrounds, or at least to the surface increase of the emplacements upon which the cells originally rested. In the bronchioles the occasional larger meshes apparently contain actual cells. Moreover, it seems to be elastic, adapting itself readily to the

slight increase and diminution of the surface of the alveolus in respiratory movements. This is not the place to discuss the biochemistry of this material, which seems to be closely allied with that between endothelial cells.

The flattened specimens of frozen sections are particularly helpful in giving us information about the silver lines. We may turn the oil immersion lens upon the spread alveolar wall and make a systematic survey of it. The lines appear of variable width, within a certain limited range. Those around the epicytes are more heavily marked. Those bounding the bare areas differ in width and density of impregnation. Some are quite well marked, and others are faint, and some even fade out altogether. Often, and particularly if the alveolus has been well distended, the line may be replaced by a succession of granules. The impression is that all the lines were originally a part of a continuous network, and that there were no free ends; but that, through the disappearance of some of the argyrophilic substance, the net has become broken in places. The lines are characterized by minor and major changes in direction, and slight inequalities in diameter are found. If the epithelial cellular material has actually disappeared from the bare areas, then we must conclude that the material of these silver lines is more resistant than the cytoplasm, and persists, adhering to the underlying connective tissue ground substance. It may become gradually attenuated and ultimately disappear.

Pores.—Pores are often seen in such material, and the edge is frequently encircled by a silver line. In such cases it is often possible to see that a line of one surface of the alveolar partition, which extends to a pore, may pass through the pore and unite with a silver line on the opposite surface of the wall; indeed more than one such union of lines belonging to opposite surfaces may occur at a pore. I have not noted this union of lines of opposite surfaces, except at pores. Such a union suggests that the pores are early in formation, and that their boundaries are originally related to the epithelial cells. Indeed, such a pore might have resulted from the dropping out of one of the pluglike epicytes, hereafter to be described, but in such a case there must be some contraction of the original cell site, because many of these pores are smaller than epithelial cells. On the other hand, some pores were found whose boundary appeared to be made up of ground substance, and here the silver line did not reach to the edge.

Often it is possible to see that the silver ring encircling an epicyte has, on the opposite side of the wall, another ring, usually of different size. In such a case the sections show that the epicyte passes right through the wall and appears as a conical plug in it. To reconcile such a formation with an epithelial cell originally on one surface only, and having a connective tissue base, it is necessary to admit a profound readjustment; but we are accustomed to having to make such readjustments in connection

with the evolution of the mammalian alveolar wall, as, for instance, in the substitution of a single capillary net for a double one, with coincidental thinning of the wall and reduction of the elastic, muscular, and connective tissue elements. The loosening and dropping out of such a pluglike epicyte (and such a falling-off is common, apparently, for epicytes which are being irritated), with appropriate adjustment of the connective tissue, might easily lead to the formation of a pore with a silver circle around it.

These flattened sections also permit of high-power study of the junctional regions already mentioned between the typical bronchiolar epithelium network and that of the adjoining alveoli. The transition from continuous epithelium to typical "respiratory" epithelium (if such it be) is often an abrupt one, the small-meshed net giving place to one of large meshes where the bare areas dominate the field. In allowing the gaze to pass along the alveolar ducts it is possible to see that the circles of myoelastic tissue forming the mouths of the alveolar ducts and sacs opening from them bear, in the case of the larger ones, the typical silver net of epithelial cells, which changes, in the smaller (seen particularly well in the muscle circles and crescents of the alveolar sacs) to a surface composed mainly of bare areas, with an occasional epicyte. One often sees these silver lines winding about the muscle rings, and also it is quite common to observe the bulging bodies of epicytes protruding from them. Capillaries frequently insinuate themselves around them, covered by the bare areas. If there is any epithelial material covering these bare areas it might be likened, in much enlarged models, to extremely thin cellophane plaques, wrapped over the bulging capillaries of the alveolar walls, and around the thinner muscle crescents, with space in between for the epicytes, and each plaque and epicyte bounded by silver lines; but when this is said, it must be realized that this hypothetical covering would be completely invisible, and such an analogy must not be taken as a close one.

Stained Preparations.—The stained preparations give us additional details. It is possible to see that the silver lines are all on the surface of the alveolar wall. Stereoscopic observation of the surface of flattened walls with high power, and study of thin sections of this material, make this clear. Never do the silver lines penetrate into the interior of the wall. If they pass from side to side, it is always through a pore. This superficial relation of the silver lines is characteristic, and differentiates them sharply from connective tissue fibers. On the other hand, it is in keeping with their recognition as originally epithelial intercellular material. This is true on the muscle-ring surfaces as well as on the surfaces of the alveolar walls. Silver lines never pass over the exposed surfaces of epicytes.

The stained preparations, too, bring out the fact that the epicytes always have nuclei, and that these are like those of the bronchiolar epi-

thelium, rounded, with large rounded nucleoli, and sparse chromatin in the resting condition. On the contrary, there are many bare areas which do not have nuclei within their borders at all, and when nuclei do appear within the confines of the silver lines, in these bare areas, they belong to the interstitial tissue of the wall and blood vessels, or to leucocytes within the capillaries. No surface nuclei in these bare areas were seen. Hence I feel that the bare areas (so far as the superficies is concerned) are without nuclei. If there were any substance in them, they would correspond to the nonnucleated squames of older authors. It seems quite evident that some of the earlier authors have mistaken the nuclei of the actual wall for the supposed nuclei of the "respiratory epithelium" in the mammals.

Stained sections, also, are useful in showing that the protoplasm of the epicytes is like that of the bronchiolar epithelium; that is to say, when the epicytes are in the resting condition. Around the nucleus there are fat globules, and a fine fuchsinophilic granulation is to be seen in the protoplasm. The cytoplasm, also, resembles that of the bronchiolar epithelium in morphology and staining properties. It should be remarked, however, that these epicytes seem to undergo changes rapidly when subjected to stimulus. For instance, under osmic acid irritation they swell up, show increase in fat globules, and definite mitotic changes in the nucleus. A discussion of their responses to irritation, however, will have to be postponed.

Nor can I go here into the details of the morphology of the wall of the alveolus except to say that, apart from the relatively small proportion occupied by the epicytes, and the much larger district pre-empted by the bare areas, overlying the capillaries (which, indeed, may prove to have no epithelial substance at all), the material of the wall is made up of mesodermal derivatives—collagenous, reticular and elastic fibers, and "ground substance." It is essentially a support for the rich network of capillaries. These tunnel through the ground substance (Macklin, 1936,[11] 1937[12]), and are supported intimately by the reticulum and indirectly by the other fibers.

I can find no foundation for the supposition that the surface silver lines are reticulum fibrils. The silver lines are fairly uniform in diameter and the pattern is quite different, as comparison with any Bielschowsky or similar preparation will show. The reticulum fibrils have a characteristic formation and blend with the collagenous fibers. They encircle the capillaries in a typical manner. Many other differences could be mentioned. It is freely admitted that too prolonged exposure to the silver will result in penetration to the deeper fibers and staining of the reticulum. Even more unlikely is confusion of the alveolar lines with collagenous or elastic fibers.

Nor is there any reason, in my opinion, for the suggestion that the silver lines represent the boundaries of endothelial cells. In my experi-

ence these are hard to demonstrate in the pulmonic capillaries of the mammal; and if they were present, their position in the wall and general pattern would separate them clearly from the surface lines. If they were endothelium lines, they should be continuous with those of the arterioles and venules—not with those of the bronchi, for instance. Moreover, the endothelium outlines are brought out by perfusion of the blood vessels, a quite different technique to that used in these experiments.

DISCUSSION OF THE EPICYTES

These plump cells, projecting with a low rounded domelike contour into the alveolar space, and showing in surface view usually a rounded oval or irregularly angulated outline, are closely encircled by prominent silver lines which appear as thin bands in sections in which the cell is cut at right angles to the alveolar surface. These silver bands are always joined to other silver lines, enclosing neighboring bare areas or other epicytes; and the network thus formed is directly continuous, in the respiratory bronchioles, with the network around the continuous epithelium of entodermal origin lining the bronchial tree, and, through the intermediation of this, with the epithelium of the pharynx which originally came from the fore-gut. Thus the epicytes and bare areas in these preparations are linked by a "silver chain" with the general entodermal epithelium. The silvered bands around the epicytes resemble those around the regular bronchiolar epithelium. They have evidently retained much of their original morphology, whereas the lines around the adjoining bare areas have become modified. The exposed surfaces of the epicytes are sprinkled with fine silvered granules, which impart a characteristic yellowish or light brownish color to them when seen in cleared sections. The absence of silver lines from the exposed surfaces of the epicytes is in keeping with the relationship which these lines bear to the bronchiolar epithelium. The epicytes lie characteristically in the capillary meshes, and may protrude through the wall as a plug; then it is common to find the opposite end also marked by a silver ring, so we have doubled rings, one on either side of the interalveolar partition. In such cases one ring is usually smaller than the other. These peglike variants of the epicyte type are seen in thin sections cut at right angles to the alveolar surface, so as to split the epicyte from end to end. The cell is really columnar in such a case. An important point is the similarity of the structure of the epicyte to that of the bronchiolar epithelium. This is seen in the rounded nucleus, rather sparsely supplied with chromatin grains, but having a prominent rounded nucleolus, and in the protoplasm with its corona of fat globules, modest in the resting phase, but prominent in the enlarged and rounded form, which is so conspicuous after stimulation, such as that of osmic acid gas. A horde of fuchsinophile granules was noted in the protoplasm. This morphologic similarity to bronchiolar epithelium reinforces our impression, derived from the

interlinkage of the silver lines, that the epicytes are survivors of the original entodermal epithelium; in this view I am in alignment with Clara[2] (1936). If the bare areas prove to be nonepithelial, as Clara[2] and others think, then these epicytes represent all that is left on the alveolar wall of the once complete layer of epithelium. The "free edge," referred to in my Durham Report,[3] would then be the irregular junction already described in this paper between the continuous epithelium of the respiratory bronchiole and the contiguous surfaces of the connecting branches and alveoli, marked by the characteristic silver lines enclosing the bare areas and epicytes. It would be difficult, perhaps impossible, to define this edge exactly. The surface transition, for instance, in the beginning of the respiratory bronchiole, is upon primitive alveolar outpocketings in which the bare areas have not fully developed. The epicytes, together with bare areas and some capillaries, are found upon the more peripheral of the muscle rings of the wall of the "respiratory" air tubes, and here, as everywhere else, they are defined by characteristic silver lines.

The epicyte readily responds to irritation by undergoing hypertrophy and dividing by mitosis, and its capacity for proliferation is great, as seen in the large numbers of offspring in inflammatory conditions. Clara[2] points out that its phagocytic potencies are different from those of the histiocytes, and he feels that this difference forms one of the arguments in favor of the view that the epicyte is not of mesodermal origin. Sprunt,[13] in my Durham Round Table Conference, stressed the fact that the exudate in the alveoli in pneumonia has two types of mononuclear cells. Bloom[14] admitted that there might be entodermal as well as mesodermal phagocytes. Although the giant cells arising from the epicytes in pathologic conditions may superficially resemble those of mesodermal origin, this circumstance constitutes no argument in itself for viewing them as mesenchymal derivatives (Clara,[2] 1936). It seems quite possible that conditions might arise which would make of this cell the nucleus of a primary cancer of the lung. Their universal occurrence seems to bespeak an important potency: I have never seen an alveolar wall without an epicyte. The entire story of their rôle has yet to be told. It is suggested that they form a reservoir for the special type of phagocyte which is said to arise from them. They seem to play an important part in the repair of the wall after inflammation. These rôles are local in character, that is, each alveolus has its own supply. In them are segregated hereditary properties which have been accumulating for millions of years.

DISCUSSION OF THE BARE AREAS

The bare areas occupy almost all of the definitive walls. In alveolar bases abutting upon arteries, veins, bronchial tubes and pleura they, as a rule, still occupy most of the territory although the proportion of epicytes has increased. They are represented on the rings and crescents

of myoelastic tissue forming the actual wall of the alveolar ducts and sacs, particularly toward the periphery of the air-duct system, where capillaries, winding over the muscle bundles underlie them. They make up the protruding capillary surfaces, and (if of epithelial protoplasm) they form the substance between the connective tissue wall of the capillary and the air (or at least the film of fluid which underlies the air—Terry[15]). They are irregularly polyhedral, and of widely varied size. Their silver-line boundaries are of variable distinctness and are often obscure or even absent. They are thinner than those around the epicytes, with which they are linked to form the same net system. In well-distended alveoli they are thinner than in those of the collapsed condition, and it is possible then to see that many of them are formed of a succession of silver-impregnated dots. In histologic preparations of normal material there is no demonstrable separate surface layer covering these areas. It seems worth noting that no separate layer has been described in normal adult mammalian alveolar walls in these areas. Faith in a continuous epithelium in the mammalian adult alveolar wall rests on evidence from pathologic or experimentally altered material. No nuclei have been demonstrated in them by the writer. What nuclei appear within their confines are definitely related to the connective tissue and blood cells. Often bare areas, clearly circumscribed by the silver lines, but in which even these mural nuclei are totally absent, are seen in cases where the capillaries have been washed out. The impression is that the bare areas, even when indefinitely bounded by attenuated or interrupted lines, represent the much enlarged loci once occupied by entodermal epithelial cells in the embryo, but from which the cells have been lost. Clara[2] suggests that this dropping off of the epithelial cells is related to the rapid growth and folding of the underlying capillaries, so that the capillary surface becomes much greater than the cell surface, leading to a breaking up of the epithelial cell layer and loss of some of the cells. Bloom[14] thinks that the epithelium of the future alveolus becomes discontinuous in fetal life, and the blood vessels push out and line the walls of the future air spaces. Palmer[16] finds the covering epithelium of the alveolus undergoing a fatty degeneration in the human embryo lung at the twenty-first week of intrauterine life, and from that time on the capillaries are nude. If the cell protoplasm has actually disappeared from these territories, it seems obvious that this boundary argyrophilic material is more durable than the cell protoplasm, and continues to adhere to the wall after the cell proper has disappeared. It is obviously capable of being stretched to accommodate itself to the much enlarged original cell emplacements. Much work remains to be done on this problem, such as the determination of age changes in the silver lines.

Again, if these bare areas have none of the original protoplasm covering them, the surface must be made up of a differentiated ground substance of the connective tissue. It must be a modified basement mem-

brane. On the other hand, if they are actually covered by extremely attenuated nonnucleated protoplasm of the original epithelial cells, then it would seem that this must be composed mainly of water, and can contain little or no stainable material; or, if stainable, then the material is so tenuous, and blends so closely with the underlying ground substance of the capillary wall, as to be invisible as a separate entity. It is noteworthy that the entire thickness of the capillary wall is less than a micron—at least in the stained preparations. Even allowing for shrinkage in technical manipulations, the wall is almost incredibly thin. Yet, with this extreme reduction to almost soap-bubble-film dimensions, it cannot be gainsaid that the capillary wall is efficient in promoting gaseous interchange, in admitting oxygen and liberating carbon dioxide and water, and also in preventing the plasma from leaking out and air from gaining access to the blood.

It seems clear that the bare areas form in association with the developing capillaries, and that they overlie the functional walls of the capillaries. The impression is that the bare areas are most extensive where the capillary net is richest. They are doubtless to be viewed as a refinement of the protoplasmic interface for gaseous exchange between the blood and air (or even between smooth muscle substance and air in the case of the smaller muscle rings) through the intermediation of the covering surface film of water, conceived of by Terry.[15] It seems likely, too, that each of the simple bare areas was originally the base of one of the embryonic epithelial cells of the primitive alveolus, though much secondary modification of this base, as well as enlargement of the area, must have occurred.

I do not think the bare areas represent filmlike extensions from the representatives of epithelial cells, the epicytes, for the epicytes are definitely circumscribed by the heavy silver lines, whereas, if they had projecting wings, it would be around these that the silver should be deposited, and no such silver-circumscribed wings have been observed. Furthermore, if the film extensions had become retracted, then we should find wrinkles around the epicytes to which they were connected, and no such wrinkles have been noted by me.

It should be said, however, that the fact that a surface film has not yet been demonstrated histologically as a definite and separate entity in unmodified normal alveolar walls of mature mammals does not prove that it is nonexistent. We know that films in tissue cultures, for instance, can be at one moment quite invisible, and at another be visible (Bremer, 1936[17]). It seems certain that the bare areas correspond to the "nonnucleated squames" of earlier authors, which were presumed to have actual substance of epithelial origin.

I regard the hypothesis of a continuous epithelial lining of the mature normal mammalian alveolar wall as not tenable, when the term "epithelium" is given its usual connotation. I am quite aware that a layer of

cells may be induced to form on the alveolar wall in conditions of inflammatory reaction, as in some osmic acid experiments which I am now conducting on living cats; but this does not prove that there is a continuous layer of epithelium there in the normal condition, and such a formation, coming as it does within the realm of pathology, is open to a different interpretation.

It must be admitted that the descriptions and interpretations of the silver lines on the mammalian alveolar walls in the literature leave something to be desired; for instance, we find it said that the silver lines cut one another in various directions, and this optical impression has been mystifying. I would suggest that it is probably due to the superposition of the two networks on opposite surfaces of an alveolar wall, when the latter is viewed "on the flat." These two networks in alveolar partitions fixed by the intrabronchial injection method, are pressed close together, and this approximation may be even closer if the wall is flattened against the slide. Proper stereoscopic observation will permit of the separation of the two networks, and what at first seemed to be a criss-cross arrangement of the lines is resolved into two networks. Again, it is possible that some workers have overexposed their walls to the silver solution, which has penetrated to, and impregnated the reticulum fibrils, and that these have intruded into the optical picture. Endothelial outlines may also have been a cause for confusion. I have given some of their distinguishing features. I would emphasize the importance of lightness in the staining with silver, applied through the bronchial tree; for confusion must inevitably arise if the silver penetrates into the substance of the connective tissue wall. These silver lines are surface formations and must be distinguished from reticulum fibrils and endothelial outlines. There are admittedly several types of linear formations which may be made to take up silver, and these must all be sharply separated from one another. I feel that the interpretation of the silver lines bounding the bare areas has been more difficult than that of those around the epicytes. That the bare area boundaries are frequently interrupted seems to be explained by the assumption that the argyrophil substance, which is their basis, disappears from the wall in places, albeit gradually and unevenly. Although each original bare area is looked upon as being the expanded base of an epithelial cell of the once continuous layer lining the alveolus, the larger bare areas are undoubtedly more or less complete fusions of two or more originally separate ones, for mergings like this must occur from local attenuation and disappearance of portions of the line system. Thus large composite bare areas of a complicated nature arise, and these may have indefinite outlines in places, and be marked by the interrupted streaks representing the silvered residue of some of the original epithelial contours.

Grateful acknowledgment of services in preparation of the sections and photomicrograph is tendered to Mr. Walter Downs.

REFERENCES

1. v. Ebner, Victor, in A. Koelliker's Handbuch der Gewebelehre des Menschen, Leipzig (Wilhelm Engelmann), 1899, pp. 304-05, Figs. 1064-66.
2. Clara, Max.: Vergleichende Histobiologie des Nierenglomerulus und der Lungenalveole, Ztschr. f. mikr. anat. Forsch. **40**: 147, 1936. (This monograph contains an excellent bibliography.)
3. Macklin, Charles C.: Report of a Round Table Conference on Pulmonic Alveolar Epithelium, J. THORACIC SURG. **6**: 82, 1936.
4. Bensley, R. D.: The Efferent Vessels of the Renal Glomeruli of Mammals as a Mechanism for the Control of Glomerular Activity and Pressure, Am. J. Anat. **44**: 146, 1929.
5. Miller, William Snow: The Lung, Springfield, 1937, Charles C. Thomas, p. 72.
6. Macklin, Charles C.: The Musculature of the Bronchi and Lungs, Physiol. Rev. **9**: 1, 1929.
7. Idem: Pulmonic Interlobular Air Passages, Proc. and Trans. Roy. Soc. Canada, Sect. V., p. 37, 1934.
8. Idem: Pulmonic Alveolar Vents, J. Anat. **69**: 188, 1935.
9. Idem: Alveolar Pores and Their Significance in the Human Lung, Arch. Path. **21**: 202, 1936.
10. Idem: Functional Aspects of Bronchial Muscle and Elastic Tissue, Arch. Surg. **19**: 1212, 1929.
11. Idem: The Mammalian Pulmonic Alveolar Wall, Elenco die Congressisti e Programma, IV Congresso Federativo Internazionale di Anatomia. Milano, Settembre, p. 13. 1936.
12. Idem: The One-Cell-Type Conception of the Definitive Mammalian Pulmonic Alveolar Wall Structure, Anat. Rec. **67**: 33, Supplement No. 3, 1937, Abst. No. 73.
13. Sprunt, Douglas H.: J. THORACIC SURG. **6**: 84, 1936. (Report of Charles C. Macklin, No. 3, above.)
14. Bloom, Wm.: J. THORACIC SURG. **6**: 83, 1936. (Report of Charles C. Macklin, No. 3, above.)
15. Terry, R. J.: Evidence of Free Fluid in the Pulmonary Alveoli, Anat. Rec. **32**: 223, 1926, Abst. No. 53.
16. Palmer, D. M.: J. THORACIC SURG. **6**: 83, 1936. (Report of Charles C. Macklin, No. 3, above.)
17. Bremer, J. L.: J. THORACIC SURG. **6**: 82, 1936. (Report of Charles C. Macklin, No. 3, above.)

EXTRAPLEURAL LOBECTOMY*

J. SEBESTYÉN, M.D.

BUDAPEST, HUNGARY

THERE is hardly any surgical intervention both the basic concepts and technical execution of which would show such marked divergence as those of lobectomy. There are two ways of performing this operation: the older and more natural transpleural method and the extrapleural method. At present American and after them English surgeons as well as a great part of the French school are endorsing intrapleural approach in one or two stages, whereas in Central Europe the influence of Sauerbruch and his school caused the extrapleural operation in more stages to become the generally accepted procedure. In order to avoid possible misunderstanding I wish to stress that I call transpleural methods all those in which the opening of the pleural cavity becomes necessary on account of whatever cause. In extrapleural operation the opening of the free pleural cavity should by all means be avoided.

On the basis of the discouraging results of one-stage interventions as performed in the past, it seemed that this type of operation will never become a suitable procedure. Practical observation, however, has shown the right way. Surgeons met the experience that if Nature has provided for adhesions in advance and if the free pleural cavity does not have to be entered, lobectomy would be a comparatively easy operation and the patients would make uneventful recovery. This has been noticed by S. Robinson as early as in 1917, and soon experimental work was started aiming at the production of artificial pleural adhesions. Many workers of reputation have studied this problem. Attempts have been started to divide the operation into several stages, and in this way operations of two and more stages were devised. Such divided operations have been studied by S. Robinson, Sauerbruch, and Zaaijer. The final elaboration and development of the procedure is credited to Sauerbruch and his collaborators. The present form of the operation took many years to develop and has gradually undergone important changes. This is due to the fact that most operations were performed for bronchiectasis and in this way the development of the technique of lobectomy is inseparably bound up with the progress of surgical treatment of this ailment. Up to 1924 surgical treatment of bronchiectasis was based on the idea that the diseased lobe must be made to shrink. This is the idea underlying the Garrè operation

Received for publication, July 20, 1937.

*From the Department of Thoracic Surgery, Dr. Joseph Vass, Industrial Hospital of the State Health Insurance Institute, Pestujhely.

("Verlagerungsmethode") with intrapleural packing. Schumacher, too, aimed at producing atrophy of the lobe by ligating its artery. Phrenicotomy, extensive thoracoplasty, and its modification with intra- or extrapleural pack compression according to Zaaijer had a similar objective. Sauerbruch, too, when he started to use extrapleural plugging between 1924 and 1926, acted along similar principles. When, however, all the procedures mentioned proved to be unsuccessful, he gradually resorted to pneumotomy, later to the Graham cautery treatment, then, after a few lobe resections, to the only radical procedure, lobectomy. It is only natural that the successive stages of development of his method clearly show the marks of the changes outlined. It would go beyond the scope of this paper and be rather out of date to give a detailed chronologic description of the variants of his method. Suffice to mention that his method has borrowed from all of the procedures mentioned. His present method probably became established during the years 1928 to 1930. On the basis of his short reports, reviews, and his manual the brief outlines of his method are the following: Preliminary phrenicotomy. First stage: exploratory thoracotomy. After the resection of a short piece of a rib, measuring a few centimeters, it is determined whether the pleural cavity is open or obliterated. Second stage: if the pleural cavity is free, introduction of an extrapleural plug. Third stage: fourteen days later, after removal of the ribs and the intercostal structures concerned, removal of the plug. Fourth stage: the isolation in several steps of the lobe to be removed. It is started along the interlobar fissure, then continued in the costovertebral angle, later from the mediastinum. Packs are inserted continually. The last step is separation of the lobe from the diaphragm. As the ease of isolation of the lobe depends on individual circumstances, the number of steps and the time intervals have to be planned accordingly. Fifth stage: after complete isolation of the root of the lobe follows the "en masse" ligation of the hilus with a rubber catheter. Sixth stage: eight to twelve days later the operation is finished with cautery removal of the necrosed lobe. The wound is treated wide open. At the Conference of the German College of Surgeons in 1934 Sauerbruch reported 58 cases operated upon in this way, with a mortality of 7 cases (12 per cent).

On this topic the recent European literature is rather scanty, so I had to build upon my own experience. In my present paper I wish to report on 16 extrapleural operations. In 14 cases the right lower lobe has been removed; in 2 cases the left lower lobe. The first cases operated upon in this way I demonstrated before the Royal Hungarian Medical Association in Budapest, November 8, 1930, as the first lobectomies performed in Hungary. At that time I followed the prescription of Sauerbruch, with paraffin-plug preparation, and I liberally

resected the thoracic wall. This operation, of course, produced a marked deformity, well shown by postoperative x-ray pictures. In 1934 I abandoned the paraffin-plug preparation and started to use sterile petrolatum gauze packs, and at the same time I contented myself with a less extensive mobilization of the thoracic wall. When comparing our present method with the former one, a considerable difference is shown in the x-ray picture, displaying itself also in a better cosmetic result. For comparison see Figs. 1 to 6. Our present method which, except for slight modifications, is essentially the same as that of Sauerbruch, can be summarized in the following points: Preliminary phrenicotomy. First stage: Hook-shaped skin incision,

Figs. 1, 2, and 3.—Removal of the right lower lobe after plug preparation, with extensive mobilization of the chest wall (Case 5).

starting paravertebrally at the level of the sixth rib and ending in the midaxillary line at the level of the tenth rib. From the seventh, eighth, and ninth ribs segments measuring 11 to 12 cm. are removed together with the intercostal structures, the resection extending medially as far as the transverse process. If in the further course of the operation difficulties are encountered in isolating the hilus, a part of the sixth rib may be removed subsequently. If the pleural cavity is closed, one can proceed at once with the mobilization of the lobe. If the pleural cavity is open, the costal pleura is exposed as extensively as possible and a sterile petrolatum gauze pack is applied. The wound is closed, save for the lowermost few centimeters. The second stage will follow after a rest period of three to four weeks. This phase consists in the mobilization of the lobe to be removed in several steps.

We prefer to attack first the diaphragmatic portion. In this phase already we identify the lower end of the interlobar fissure, which we mark with catgut stitches or with a gauze pack in order to facilitate its finding again at the next occasion. If the pleural cavity is open, we finish the first stage with dissecting free the diaphragmatic part.

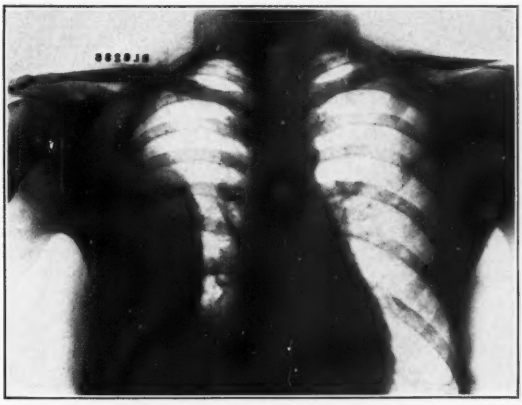


Fig. 2.

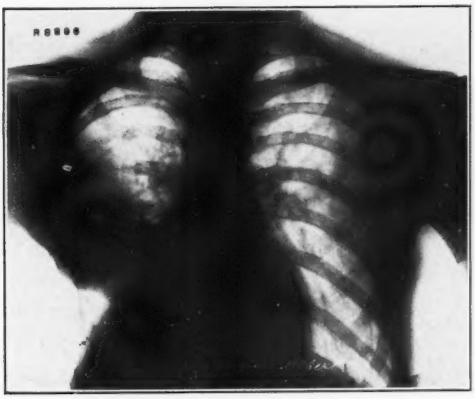
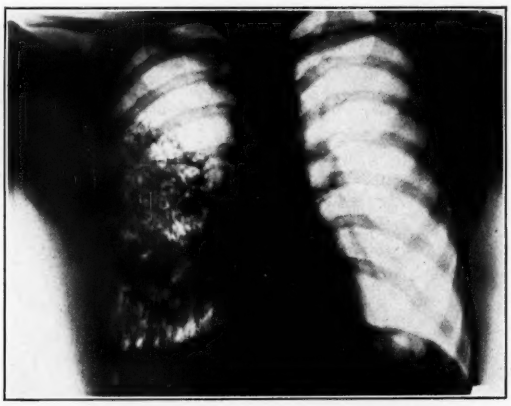


Fig. 3.

In the second stage the isolation is carefully continued in the costo-vertebral angle and the pulmonary ligament is cut. The dissected parts of the lobe must be carefully packed in order to prevent the formation of fresh adhesions. In the third stage we enter chiefly the interlobar fissure and try to isolate the lobe to be removed from its neighbor. The time interval between the individual steps is eight to ten or twelve days, according to the case. The isolation of the lobe is

performed, if technically possible, extrapleurally, i.e., the lung is mobilized with both the parietal and visceral pleurae adhering to it. Often this is impracticable throughout, and in a later stage one often must carry out the dissection between both layers. If the pleural cavity is closed, this will have no ill effects. In the third stage, after



Figs. 4, 5, and 6.—Removal of the right lower lobe after petrolatum gauze pack preparation, with moderate mobilization of the chest wall. Good cosmetic result (Case 11).

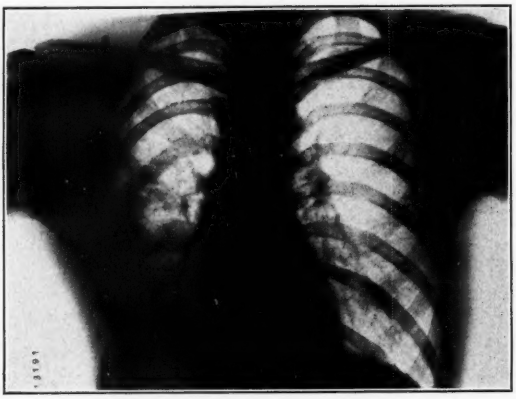


Fig. 5.

more complete isolation of the hilar region, it will become possible to tighten a thin rubber catheter around the hilus. The catheter is transfixed distally with a strong silk suture in order to prevent its slipping. The fourth stage consists of cautery removal of the necrosed lobe. This is usually performed in the third to fourth day after the constricting catheter has been applied. The wound is treated wide-open and its healing occurs by secondary granulation. The removal of the lobe is

usually performed under local anesthesia and but exceptionally, if the patient is uncooperative, under superficial ether inhalation narcosis. Of course the procedure outlined should serve but as a pattern and departures from it will often be necessary. If there is the slightest suspicion that the pleural cavity is not completely obliterated, one should proceed with still greater care, and it often will be advisable to increase the number of successive steps. Conversely, if there are favorable adhesions, quicker work is possible. As it can be seen, it is difficult to give a standard description of the procedure, for it has to be modified according to the individual case. Both the number of

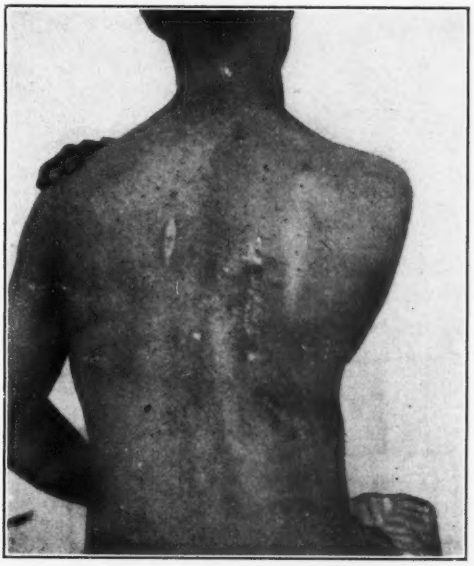


Fig. 6.

stages and the time intervals between them will depend on the given conditions.

If the pleural cavity is closed, the method is simplified and there is no need for a preparatory operation. Under favorable conditions the entire operation can be carried out in two stages, and the ligature of the hilus may be possible as early as in the second stage. These cases, however, are exceptional, the pleural cavity being in most cases at least partially open. Under such circumstances the preparatory operation becomes an important step. As mentioned, at first we proceeded according to the method of Sauerbruch and used the paraffin-plug method. The plug is introduced through the hole of a sparing rib resection, through which the costal pleura must be freed over the

Fig. 7.—Bronchiectasis in right lower lobe (Case 13).

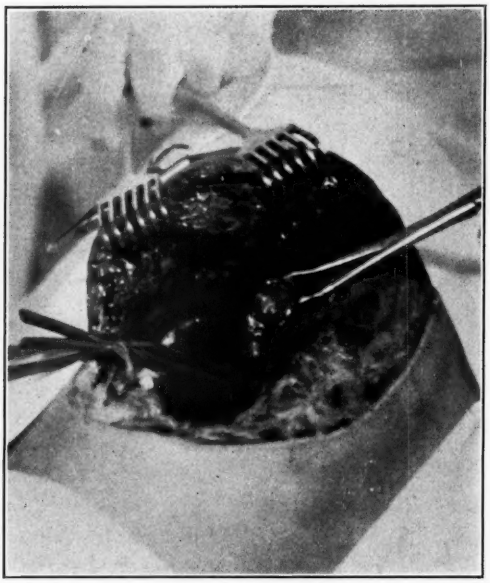


Fig. 8.—The isolated lobe is ligated.

entire lobe to be removed, if possible. The extrapleural cavity produced in this way is now filled with 400 to 600 gm. of Baer's paste. This can be usually accomplished if the pleural cavity is open and the

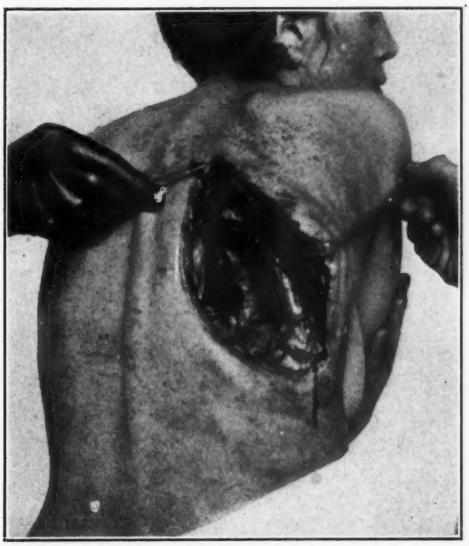


Fig. 9.—Wound conditions immediately after removal of the lobe.

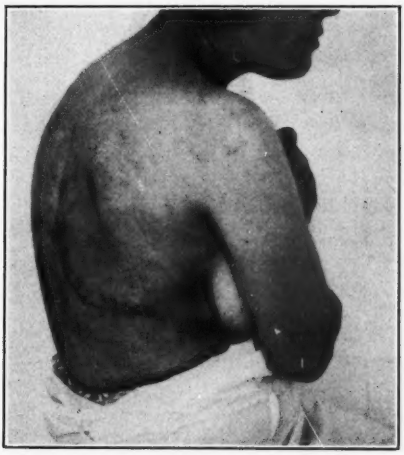


Fig. 10.—The same patient after recovery.

pleura thickened. If, however, the pleura is thin and adherent, there is a serious danger of injury. Even the mere introduction of the extrapleural plug through the small hole cannot be considered an

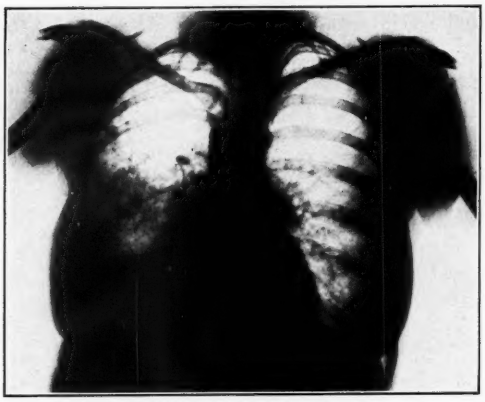
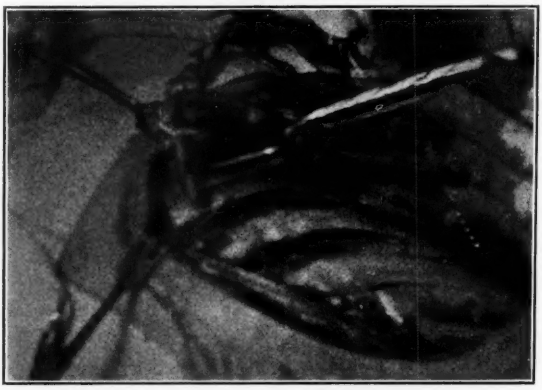


Fig. 11.—X-ray picture after lobectomy (Case 13). (Petrolatum gauze pack preparation.) Some residual iodized oil is seen.



Figs. 12-16.—Several phases of the extrapleural lobectomy.
Fig. 12.—Shortening of the seventh, eighth, and ninth ribs.

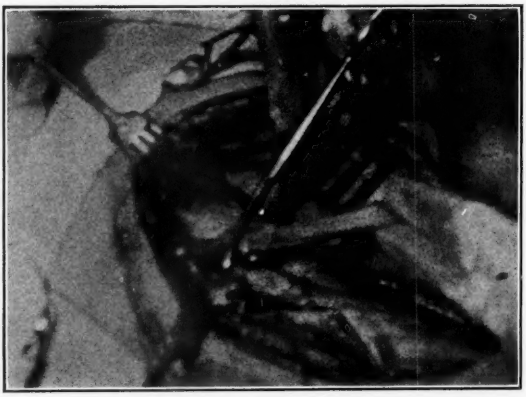


Fig. 13.—Removal of intercostal structures.

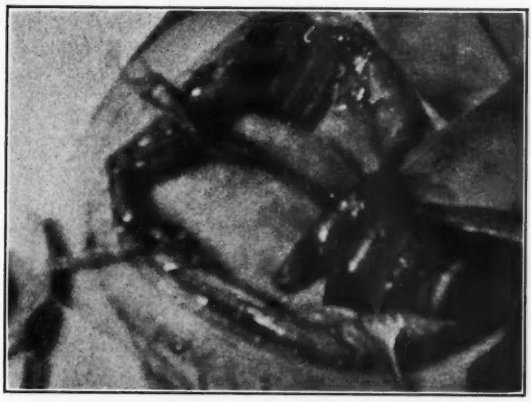


Fig. 14.—Extrapleural isolation of the lobe.

Fig. 15.—Mass ligation of the lobe.

Fig. 16.—Cautery removal of the lobe. In this case gauze pack preparation was used.

intervention devoid of danger. Though I did not experience immediate danger from this intervention when preparing for lobectomy, I had cases of injury to the pleura by the plug in operations for lung abscess and other pulmonary conditions. After the introduction of the plug we used to wait at least three to four weeks, and yet on several occasions we entered the free pleural cavity when trying to isolate the lobe after removal of the plug. I absolutely agree with J. Alexander* who is sceptic about the ability of the plug to produce mediastinal and hilar adhesions. In fact, the greatest shortcoming of the procedure is that the plug will produce adhesions, even after long periods of waiting, only at the sites where it is in immediate contact with the pleura. If one goes beyond this area when attempting to isolate the lobe, entering the pleural cavity may easily occur. Often open pleural cavity and exudate are found instead of adhesions, in spite of massive scarring around the plug. Under favorable circumstances the patient will get a partial pneumothorax and this is but an annoying accident. The operation has to be terminated at once, and by applying high pressure or after aspirating the pneumothorax the rent is to be repaired. If this be impossible, packing is indicated. In most cases after waiting a few weeks the operation can be continued. If, however, the pneumothorax thus produced is complete, serious shock will result, and the pneumothorax must be considered infected at once, as the wound has been open for several days or even weeks, according to the phase in which this mishap occurred. Such cases of ours as a rule terminated fatally, and of our four operative deaths two were due to pneumothorax complication. The earlier the pack is removed, the more liable this complication is to occur. Conversely, if the plug is left in place too long, it will become surrounded by massive scar tissue, a so-called "plug bed." Orientation and chiefly the finding of the interlobar fissure may be made extremely difficult by it. Of both methods of preparation, I think the petrolatum gauze pack method is more advantageous, considering the fact that it causes less irritation and, therefore, after its use orientation is easier. In preparing for the insertion of the pack, injury to the pleura can be positively avoided. In the further phases of the operation, however, the danger of producing pneumothorax is just as possible as with the plug method. For those preferring the plug technique the Hertel† method seems recommendable, as with this—though it has other drawbacks—injury to the pleura can be more effectively avoided. I always start isolation of the lobe from the diaphragmatic surface, because basal adhesions are almost invariably present, even if the rest of the pleural cavity

*Surg., Gynec. & Obst. 56: 658, 1933.
†Chirurg. 6: 95, 1934.

be free. In this way injury to the pleura is less likely to occur, as the irritation of the gauze pack will provoke ever progressive adhesions. When the isolation has advanced to the upper part, adhesions will be present there. We observed the occurrence of a partial pneumothorax when dissecting near the upper end of the interlobar fissure and in the hilar region. We experienced no disadvantage whatever from starting dissection at the diaphragm. There are surgeons who prefer first approaching the interlobar portion, because there is stagnation of secretions in the lobe separated from the diaphragm. Anyhow, the condition of the patient becomes increasingly precarious as the isolation of the lobe becomes more complete. In the later stages of the operation the lobe is unable to get rid of its secretions, the heart muscle has also suffered, therefore, it is advisable to keep shorter intervals between the steps of the intervention and to hurry its termination.

As to aftercare, there is hardly anything extraordinary to mention. Cases operated upon according to the method described will invariably heal with a bronchial fistula, needing later closure. After healing of the wound, the fistula can be closed with a minor plastic operation, using a muscle flap according to the principles of Lebsche. During the last few years I have not waited until the wound becomes filled up completely, but I have tried to close the fistula earlier. There generally remains some connective tissue around the hilus, sufficient to allow covering of the tied bronchial stump. This early closure of the fistula effects a considerable shortening of the period of recovery. Our cases treated in this way were cured in four to four and a half months.

As to the etiology, pathology, and symptomatology of bronchiectasis, the American literature is so replete with excellent treatises on this subject that I consider superfluous to touch on it. Surgical treatment is influenced by two factors: the underlying pathology and the clinical behavior of the case. For the surgeon it is important to know that isolated bronchiectasis of one lobe is a rare condition. Bronchiectasis cannot be considered a local disease, and this circumstance is a disturbing factor in its operative treatment. In determining the operative indication it should be borne in mind that diagnostic errors are liable to occur in spite of the most conscientious examination. We had a case in which the right middle lobe was mistaken for the lower one. According to our experience, the coexistence of a seriously diseased lower lobe with a completely healthy middle lobe is an exceptional occurrence. In such cases an x-ray picture taken obliquely will be often useful. We had, however, cases in which slight changes of the middle lobe became observable only after removal of the lower

TABLE I

NUMBER OF CASES	AGE AND SEX	ETIOLOGY	YEARS DURATION OF DISEASE	GENERAL CONDITION	QUALITY AND QUANTITY EXPECTORATION	LOCALIZATION	DATE PREPARATORY OPERATION	METHOD PREPARATORY OPERATION	METHOD OF OPERATION	OPERATIVE DEATH	SPUTUM	REMARKS AND COMPLICATIONS
1. F. Gy.	23 M.	1917 pleuritis	13	Bad	150-200 c.c. fetid	Right middle and lower lobes	1/13/30	Paraffin plug	Lobectomy and cauterization Graham	—	30-50 c.c.	6/4/34 Death through bronchiectasis
2. Sz. T.	24 F.	1925 pneumonia	5	Good	Temporary 200 c.c. mucopurulent	Right lower lobe	2/26/30	Paraffin plug	Lobectomy	—	—	—
3. S. Gy.	25 M.	1926 pleuritis	4	Good	150 c.c. fetid	Right lower and some in r. upper lobe	3/10/30	Paraffin plug	Lobectomy	—	10-20 c.c.	Partial pth. during op.
4. F. T.	37 M.	1929 acute beginning pneumonia?	1	Bad	400 c.c. mucopurulent	Right lower lobe	4/2 /31	Paraffin plug	Lobectomy	—	10-20 c.c.	Often hemorrhages
5. Sz. A.	25 M.	Uncertain	Since childhood	Good	200-300 c.c. purulent	Right lower lobe	7/ 4/31	Paraffin plug	Lobectomy	—	—	Clubbed finger
6. L. T.	44 F.	Uncertain	2	Satisfactory	100-150 c.c. fetid	Right lower lobe	6/16/32	Paraffin plug	Lobectomy	7/27/32	—	Thrombosis of the pulmonary artery
7. G. T.	25 M.	1930 influenza, pleuritis	3	Good	150-250 c.c. fetid	Right lower lobe	5/ 2/33	Paraffin plug	—	6/3/33	—	Open pth. aspiration-broncho-pneumonia. Acute sepsis

TABLE I—CONT'D

8. W. A.	28 M.	1918 influenza, pneumonia, pleuritis	7	Good	100-150 c.c. fetid	Right lower and middle lobes	11/ 7/33	Petrolatum gauze pack	Lobectomy	—	30-40 c.c.	Partial pth. during op. Clubbed finger
9. K. T.	40 F.	Uncertain	Uncertain	Satisfactory	250-500 c.c. fetid	Right lower lobe	12/ 4/33	Petrolatum gauze pack	—	12/24/33	—	Preceding pth. treatment. Open pth. Sepsis
10. T. Gy.	31 M.	1925 ascertained	8	Bad	180-200 c.c. fetid	Right lower and middle lobes	12/29/33	Petrolatum gauze pack	Lobectomy and cauterization Graham	—	15-20 c.c.	In 1925 operation in narcose (Stomach perforation)
11. G. B.	22 M.	1932 acute hemoptysis	2	Good	150 c.c. fetid	Right lower and middle lobes	1/15/34	Petrolatum gauze pack	Lobectomy and cauterization Graham	—	30-40 c.c.	During cauterization air embolism
12. P. T.	37 F.	1918 and 1929 pneumonia	5	Satisfactory	100-150 c.c. mucopurulent	Left lower lobe	5/28/34	Petrolatum gauze pack	Lobectomy	—	—	Partial pth. during op. Preceding closure of the fistula
13. P. F.	26 F.	Pneumonia in childhood	Since childhood	Good	50 c.c. temporary 200 c.c. purulent	Right lower lobe	12/21/34	Petrolatum gauze pack	Lobectomy	—	—	Preceding closure of the fistula
14. N. S.	40 F.	1911 pneumonia	6	Bad	150 c.c. fetid	Right lower lobe	3/27/35	Petrolatum gauze pack	Lobectomy	—	—	Partial pth. during op. Preceding closure of the fistula
15. M. G.	20 M.	Uncertain	1½	Good	50-150 c.c. fetid	Right lower lobe	8/10/35	Petrolatum gauze pack	Lobectomy	—	—	Preceding closure of the fistula
16. Sz. T.	15 M.	1932 empyema necessitatis	3	Satisfactory	100-130 c.c. fetid	Left lower lobe	12/16/35		Lobectomy	12/24/35	—	Clubbed finger. Purulent pericarditis. Sepsis

lobe. If the pathologic changes of the middle lobe are so slight as to cause the expectoration of not more than 2 to 3 gm. of residual sputum, this is practically negligible, and such cases are taken for cured in my tabulation of cases. Unfortunately, if the wound is treated wide-open, the residual sputum will make its appearance only after the closure of the wound and of the bronchus. During the interval between the tying off and the removal of the lobe, symptoms of a residual bronchiectasis may be silent, and later the mucopurulent secretions will escape through the bronchial fistula and become irrecognizably mixed up with the wound secretions. In this respect the extrapleural method is not satisfactory. In no case presenting serious symptoms due to middle lobe involvement after removal of the lower lobe have I been able to remove the diseased middle lobe later. The wide-open treatment causes such extensive scarring that it is impossible to make out the confines of the middle lobe. In such cases I had to content myself with partial removal of the middle lobe, or I treated it according to the Graham method. In the course of cauterizations near the hilus, however, I was greatly annoyed by occasional air embolism and secondary bleeding. In cases treated in this way I never succeeded in obtaining complete freedom from symptoms. We had one diagnostic error in mistaking the right lower lobe for the middle one, and in another case changes in the lower portion of the left upper lobe were masked by extensive changes of the left lower lobe. In no case did x-ray pictures taken obliquely yield sufficient information.

The second factor playing a part in the surgical treatment, but chiefly in determining the operative indication, is the clinical behavior of the individual case. The protean clinical manifestations of bronchiectasis are well known. Cases ranging from the mildest form to the most serious septic one are observed. Not only the degree of severity, but also the course is variable. There may be a quiescence of symptoms for years, symptoms appearing only at intervals. It is almost impossible to make a prognosis as to the future development of the disease, therefore, the problem of operative indication is a delicate one. In milder cases, as their future going downhill is by no means certain, it is wiser to choose expectant treatment. Of course the result of operative treatment will be more favorable if only mild cases are operated upon, whatever method be adopted, whereas severe cases are liable to make the statistics worse.

My 16 cases are summarized in Table I. The case histories of my 4 fatalities, as they are rather instructive, will be given in detail. They are the sixth, seventh, ninth, and sixteenth cases, respectively, of the series.

CASE 1.—L. J., female, aged forty-one years. Her complaints dated back for years. For two years she had expectoration. Since February, 1932, her state was rapidly declining. She had the daily amount of 100 to 150 c.c. of foul smelling sputum. She had periods of fever. Sedimentation speed was 22 mm. in one hour, 40 mm. in two hours. Colloid lability test 2+. Body weight 63.15 kg. General state of health was satisfactory. At x-ray examination in the right lower lobe typical sacciform bronchiectasis was visualized. Preparatory operation by the plug method was performed on June 16, 1932. The thoracic conditions were rather favorable, so that the lobe could be ligated on July 24, 1932. The intervention was well tolerated by the patient. Two days later, however, at 6 A.M. she suddenly felt sick and died under signs of progressive dyspnea and cyanosis at 2 P.M. In our opinion symptoms pointed to the death being due to a slow, progressive form of thrombosis of the pulmonary artery, originating in the ligated hilus. Our presumption was confirmed by findings of autopsy, which at the same time cast light on other, unsuspected conditions. It turned out that at the operation the greatly dilated middle lobe had been isolated and tied. The severely sclerosed bronchiectatic lower lobe was left in the costovertebral angle as a scarry layer, measuring about half an inch in thickness. This degenerated lobe was so distorted and shrunken that its recognition was difficult even at autopsy. It contained no healthy lung tissue but was converted into a mass of scar tissue containing cavities. It was so firmly adherent that it had to be dissected away with considerable difficulty. The wound was found to be in order. The healthy middle lobe completely occupied the place of the lower lobe. The preparation for the plug interfered with clear orientation; however, the extraordinary severity of pathologic changes precluded the possibility of successful surgical intervention.

CASE 2.—G. J., male, aged twenty-five years. In January, 1930, he had had influenza with pleurisy. Since that time, he expectorated daily 150 to 250 c.c. of foul smelling sputum. He had periods of fever. Sedimentation speed was 32 mm. in one hour, 66 mm. in two hours. Colloid lability test 3+. Weight 60.50 kg. Good general health. X-ray examination showed typical, extensive bronchiectasis in the right lower lobe. Preparatory operation with paraffin plug on May 2, 1933. Four weeks later, on May 31, we removed the plug, and as the pleural cavity seemed to be closed, we at once proceeded with the mobilization of the lobe at its diaphragmatic part. Scarcely did we advance with the mobilization a few centimeters, when the pleura opened and an open pneumothorax resulted. We had to dissect through a sclerosed layer as thick as half a centimeter, behind which we found the free pleural cavity with exudate. Signs of severe circulatory embarrassment at once appeared, forcing us to use high pressure and to quickly complete the operation. The patient developed progressive circulatory failure and difficulty in expectoration. He died on June 3, with symptoms of acute sepsis. Autopsy revealed besides slight serous pleurisy of the operated side severe aspiration bronchopneumonia of the contralateral lung. The other organs showed septic changes. This case clearly demonstrates the possibility of finding unobliterated pleural cavity even after a four weeks' preparation with a paraffin plug. This serious complication is the more prone to occur, the earlier the plug is removed.

CASE 3.—K. T., female, aged forty years. There are no reliable anamnestic data. A year ago she had been treated with artificial pneumothorax for bronchiectasis. This treatment, however, having proved inefficacious, was discontinued in November, 1932. Since June, 1933, her condition had grown progressively worse. The daily amount of her sputum rose to 250 to 500 c.c. She lost 14 kg. On admission her sedimentation speed was 44 mm. in one hour, 82 mm. in two hours. Colloid lability test 4+. Body weight 51.40 kg. She had temperatures up to 38° C. Her general state

of health was satisfactory. X-ray examination visualized bronchiectasis of both the cylindrical and sacciform type in the right lower lobe. Preparatory operation was performed by the pack method on December 4, 1933. Seven days after this intervention the patient suddenly felt very sick, so that I was forced to revise the wound. When changing the dressing, I noticed that a palm-sized area of the costal pleura separated at the preparatory operation was necrotic, and presently was extruded together with the pack. The patient contracted an open pneumothorax and a consecutive sepsis to which she succumbed on December 24, 1933. This unusual complication must be explained by some residual pneumothorax left over from the previous treatment, though it was not visualized on x-ray examination. The thoracic cage becomes somewhat narrowed by the preliminary operation and in this way the extensively dissected costal pleura, on account of the residual pneumothorax, had no immediate contact with the visceral pleura. Pressure and lack of nutrition caused its necrosis.

CASE 4.—Sz. I., male, aged fifteen years. In 1932 he had had an empyema necessitatis. For the last three years he had the daily amount of 100 to 130 c.c. of foul smelling sputum. There was clubbing of the fingers. Sedimentation speed 60 mm. in one hour, 82 mm. in two hours. Body weight 45 kg. He had temperatures ranging up to 39° C. His general state of health was satisfactory. X-ray examination showed, aside from considerable thickening of the pleura, typical bronchiectasis of the left lower lobe, especially in the costovertebral angle. At operation on December 16, 1935, the pleural cavity was found to be obliterated, therefore, we proceeded at once with the isolation of the left lower lobe. The anatomic situation was so favorable that three days later we were able to ligate the hilus, and on December 21 we removed the lobe with the actual cautery. The patient died on December 24, 1935. Autopsy showed purulent pericarditis and mild bronchopneumonia of the right lower lobe. This case is a warning against too quick succession of stages and progress of isolation. The surgeon should resist seduction by favorable thoracic conditions. The organism needs time to develop its defense mechanism.

Under the influence of the American literature in 1935, I performed my first one-stage transpleural lobectomy. The total number of my cases performed according to other than the extrapleural method is but 4. All operations were performed under infiltration anesthesia combined with morphine scopolamine preliminary medication. All operations were done for bronchiectasis, after the prescribed preliminary treatment. In three patients I performed the operation according to the suggestion of Brunn. Of these, 2 recovered and 1 died. One patient operated upon according to the Nissen* method died in consequence of the operation. The first patient operated upon according to Brunn died on the tenth postoperative day. At autopsy the suture of the hilus stump was found to be airtight, the remaining left upper lobe was found to be collapsed. The failure of the remaining lobe to expand was due to pathologic changes in its lower portion. Superimposed changes of the lower lobe covered dilatations in the lower portion of the left upper lobe. This case supports the opinion of Brunn about the fate of one-stage operations: this does not depend on

*Deutsche Ztschr. f. Chir. 247: 289, 1936.

the tightness of the stump, but on the ability of the remaining lobe to expand. Moreover, his opinion is corroborated by two cases which recovered; in both cases the bronchus opened up a few days after operation and a basal empyema developed, after which both went on to eventual recovery. In one-stage operations I consider the preliminary pneumothorax to be of great importance. Aside from its allowing some preliminary orientation concerning the thoracic status, it facilitates the adaptation of the organism to the lack of the organ. If the lung is collapsed, the danger of aspiration is minimized; it is much easier to secure the hilus and the surgical shock, too, is less severe. There has not been and even at present there is no safe procedure for closing the bronchus. An infected stump, closed by whatever method, is liable to break down. The death of the patient operated upon according to the method of Nissen was due to a technical fault. Probably I drew the constricting catheter too tight when I first applied it and circulatory disturbances in the lobe to be removed started before the remaining lobe could expand and get fixed by adhesions, so the field of operation failed to become walled off.

I am not going now to criticize the one-stage method, the number of my cases being too small. On the basis of my limited experience, however, I got the impression that the success of this operation is subject to many factors, entirely independent of the surgeon. Unexpected technical difficulties may arise, greatly influencing the result. For instance, in one-stage operation the structures of the hilus have to be secured separately. This may be, on account of unforeseen causes, impossible or not completely possible. If, however, it must be secured en masse, the dangers resulting from opening of the bronchus are increased. *Whereas in the removal of tumors the one-stage operation will unquestionably become the operation of choice, in bronchiectasis individual consideration of each case seems preferable.* Of course, the extrapleural method is not devoid of danger either. It taxes the endurance both of surgeon and patient; its immediate hazard, however, is decidedly slighter.

My transpleural operations were the following:

CASE 1.—N. K., male, aged twenty-three years. His illness started in 1929 with pneumonia. During the year 1932 his condition grew progressively worse. He expectorated the daily amount of 160 c.c. of foul smelling sputum. On admission his sedimentation speed was 38 mm. in one hour, 63 mm. in two hours. Colloid lability test 2+. Body weight 63 kg. There was no fever. The patient enjoyed excellent general health. X-ray examination disclosed extensive bronchiectasis of the left lower lobe. After several weeks of preliminary artificial pneumothorax treatment and phrenicotomy and adequate preoperative management, I performed the one-stage operation, according to the prescription of Brunn on February 26, 1935, under infiltration anesthesia with preliminary morphine-scopolamine medication. Sundering the lobes was not difficult, and the structures of the hilus were secured separately with ease. The entire operation was performed without loss of blood. When

inspecting the upper lobe, however, it could be noticed that the lower portion of the upper lobe showed pathologic changes; at places there were scattered retracted scars. The patient left the operating room in excellent condition. However, it soon became apparent that the patient continued to void sputum of the previous type. Through the rubber tube an increasingly purulent, later foul smelling secretion drained. The temperature became septic. On the sixth postoperative day the patient became acutely ill and complained of stabbing pains in the opposite side of the chest. He died under signs of increasing respiratory embarrassment and septicemia on March 6, 1935. Autopsy showed the left upper lobe to be atelectatic and completely collapsed. In its lower portion there was typical bronchiectasis. The suture of the hilar stump was competent. There was purulent pleuritis on the operated side, with involvement of the mediastinum and the contralateral pleural cavity. The latter contained 600 c.c. of seropurulent fluid. In the right lower lobe there were foci of early bronchopneumonia. The other organs showed signs of sepsis. The

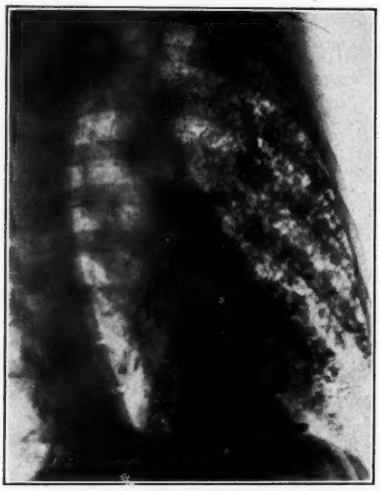


Fig. 17.—Bronchiectasis in left lower lobe (Case 2).

tragical issue in this case was due to a diagnostic error. It can happen in spite of the most careful examination that changes in the lower portion of the left upper lobe being covered by extensive changes in the lower lobe will escape detection. Though the closure of the stump was tight, the remaining diseased lobe was unable to expand. This circumstance contributed to the development of empyema. As mentioned, this case supports the view of Brunn, according to which the fate of a one-stage operation does not depend on good closure of the bronchial stump but on the ability of the remaining lobe to expand. If on account of whatever cause the remaining lobe fails to expand and to get fixed by adhesions within a few days, serious complications are to be feared.

CASE 2.—L. I., male, aged twenty-four years. In January, 1933, he had pertussis. Since that time he is expectorating the daily amount of 160 to 180 c.c. of foul smelling sputum. On admission the sedimentation speed was 35 mm. in one hour, 61 mm. in two hours. Colloid lability test 3+. Body weight 57.50 kg. At times he had temperatures up to 38 to 38.5° C. X-ray examination disclosed typical bronchiectasis

in the left lower lobe, situated chiefly posteriorly. After adequate preliminary management, I performed the one-stage lobectomy according to the Brunn method on January 28, 1936. At operation extensive adhesions were found between the upper lobe and the upper portion of the lower lobe. Perihilar adhesions made secur-

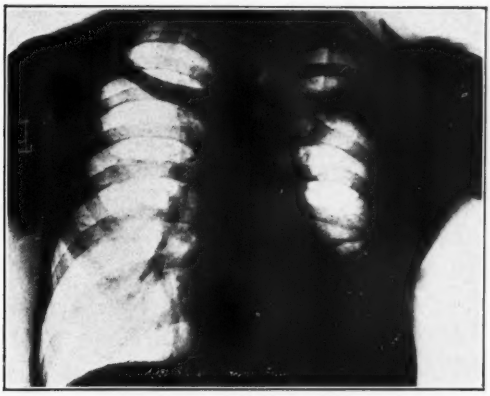


Fig. 18.—One-stage removal of left lower lobe according to the method of Brunn.

Fig. 19.—Check-up x-ray picture of the remaining left upper lobe. There is still some residual pneumothorax at the apex.

ing of the structures of the hilus difficult. After severing some bands binding down the upper lobe I closed the incision. According to prescription I instituted airtight drainage of the pleural cavity. The patient was summoned up several times to remove by coughing the residual air from the chest. Its bubbling through the

tube end placed under water was checked. For a few days blood-tinged serum drained through the tube, later it progressively became more cellular in character. On February 22 it became evident that the bronchus was open. The discharge became purulent. We started irrigation of the thoracic cavity with azochloramide solution. In the eighth postoperative week the rubber tube could be removed, and spontaneous closure of the thoracic fistula ensued. The entire postoperative course was characterized by subfebrility. After another three to four weeks, the patient recommenced to have sputum and again became febrile. A new fistula formed in the wound of the thoracotomy, from which foul smelling pus discharged. After closure of the fistula, the patient became symptom free for several weeks, but soon the same troubles reappeared. Recurrences followed at intervals of from three to four weeks. Fluid injected into the fistula appeared in the sputum, demonstrating the presence of a bronchial fistula. We treated the fistula with a 40 per cent silver nitrate solution. This condition persisted for about four months. At the end of July, 1936, the fistula permanently closed; at its site the skin retracted and became pigmented. The patient feels cured.

CASE 3.—H. R., aged thirty-five years. His illnesss started acutely in 1930 (probably pneumonia or pleurisy). He is expectorating the daily amount of 200 c.c. of mucopurulent sputum. Sedimentation speed is 18 mm. in one hour, 46 mm. in two hours. Colloid lability test 2+. Body weight 56.80 kg. The patient has no fever; his general condition is excellent. X-ray examination disclosed typical sacciform bronchiectasis of the left lower lobe, situated chiefly in the costovertebral angle. As I decided to do a one-stage lobectomy, I first wished to institute a preliminary artificial pneumothorax treatment. This, however, proved to be unfeasible. On x-ray examination some air was visible in the pleural cavity, but the patient complained of distress, and as after each repeated attempt to introduce more air there was a febrile reaction, we discontinued further experimentation. Presuming the presence of extensive pleural adhesions, I gave up the plan of one-stage transpleural operation. On September 26, 1936, according to the extrapleural method, I resected the seventh, eighth, and ninth ribs paravertebrally, together with the intercostal structures. In the belief that the pleural cavity was obliterated I at once proceeded with the isolation of the lobe. However, to my greatest surprise I entered the free pleural cavity, and after a few thin pleural adhesions gave way, I saw the completely collapsed left lung. In this emergency situation it occurred to me to resort to the method recently described by Nissen, who used it when he faced a similar situation after plug preparation. I placed a rubber catheter around the hilus, ready for being tied, surrounded the lobe with gauze packs, and closed the wound partially. The removal of the lobe could still be accomplished, but the patient died of sepsis on October 3, 1936. Autopsy disclosed suppurative pleurisy, fibrinous pericarditis, and myocardial degeneration. Aside from an accessory left lobe, the collapsed left upper lobe showed no other pathologic change. The disaster was due to the appearance of circulatory troubles in the ligated lobe before the upper lobe had time to expand and before the field of operation could be walled off by protecting adhesions.

CASE 4.—S. J., female, aged twenty-three years. Her symptoms date back to childhood. Since February, 1935, her condition is rapidly declining. She has the daily amount of 200 to 300 c.c. of foul smelling sputum; her breath is offensive. Sedimentation speed 28 mm. in one hour, 42 mm. in two hours. Colloid lability test 2+. At times she has fever. Body weight 62.50 kg. Her general condition is good. X-ray examination disclosed sacciform dilatations involving the entire left lower lobe. Operation according to the method of Brunn was performed on February

17, 1937, after the usual preoperative management. The pleural cavity was found to be completely free, and the lower lobe showed no gross pathologic change. No difficulty was encountered in securing the structures of the hilus separately. The immediate postoperative course was uneventful. On the twelfth day unequivocal signs of opening up of the bronchial stump made their appearance. The discharge became purulent. The condition of the patient was satisfactory throughout. Spontaneous closure of the fistula ensued in April, 1937.

SUMMARY

Sixteen consecutive extrapleural lobectomies are reported. In 14 cases the right lower lobe, and in 2 cases the left lower lobe was removed. Of the cases mentioned 1 patient remained unimproved, 5 patients improved, and 6 completely recovered. There were 4 fatalities in the series, 2 of which were due to pneumothorax complication, 1 to thrombosis of the pulmonary artery, and 1 to suppurative pericarditis. Of another 3 patients operated upon, according to the technique of Brunn, 2 recovered, 1 died. In this latter case death was due to pathologic changes in the left upper lobe which escaped detection. One patient operated upon according to Nissen died. The latter 4 cases were left sided. All of the 20 operations were performed for bronchiectasis. After a brief discussion of the merits of both the extrapleural and intrapleural methods, the author comes to the conclusion that in the surgical treatment of bronchiectasis the extrapleural method must also be classed among the procedures to be considered.

NOTE.—Since the preparation of this article another patient has been successfully operated upon, by the method of Brunn.

In Memoriam

HANS CHRISTIAN JACOBAEUS

1879—1937

The death of Hans Christian Jacobaeus, Honorary Member of the American Association for Thoracic Surgery occurred on October 29, 1937. He was born on May 29, 1879, in Scania, a southerly province of Sweden. Educated in the ancient universities of Lund and Stockholm, Jacobaeus rose to the eminent position of Professor of Medicine at the Caroline Institute, and head of its Second Medical Clinic. He was a member of the Royal Medical Board and from 1925 Chairman of the Nobel Committee of the Caroline Institute.

After early work in the clinical and pathologic laboratories Jacobaeus interested himself in the subject that was to bring him world fame. He started with the cystoscope to inspect disease processes in the pleural and peritoneal cavities, publishing his observations in 1910. Having actually seen the pleural adhesions that prevented an adequate collapse of the lung by artificial pneumothorax he next worked out his method for severing them with the galvanocautery. With Hj. Tideström he published in 1914 the first paper on closed pneumolysis, "A New Method of Removing Adhesions in the Pneumothorax Treatment of Pulmonary Tuberculosis."

In later publications Jacobaeus described the technique and indications for his operation with such thoroughness that only minor modifications have been added by others. Closed pneumolysis was quickly adopted by the medical world and stands today as an operation of unquestioned value in the treatment of tuberculosis.

In recent years Jacobaeus and his pupil Björkman worked actively to perfect a method for determining the function of each lung separately. His scholarly paper on this subject with its far reaching physiologic and clinical applications awakened the interest and enthusiasm of our Association at the 1937 annual meeting.

While foreign countries may honor Jacobaeus as scholar and scientist, the touching tributes of his Swedish colleagues bear witness to the love and affection his countrymen held for Jacobaeus the physician and man.

Edward D. Churchill.